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ARCHIVES OF OPHTHALMOLOGY.

THE TREATMENT OF THE IRIS IN CATARACT OPERATIONS.

BY LT. COL. HENRY SMITH, C.I.E., I.M.S. (RETD.).

THIS is a matter which is inadequately treated in writings on ophthalmology, being passed over as unimportant, while it is the reverse.

I must first consider the various procedures—Czermak's operation, the stitching of conjunctival flaps, corneal sutures —which have been advocated as a means of preventing prolapse of the intact iris and of getting away from an iridectomy. They have no other *raison d'être*.

My experience of them has been very far from favorable.

In Czermak's operation the conjunctival wound is healed long before sclero-corneal union is sound—with the reëstablishment of tension aqueous props the sclero-corneal wound open. Prolapse into this space frequently occurs though it is not apparent to casual observation, and is the worst form of prolapse, for it includes the base of the iris. It leaves a very irritable eye liable to trouble of a sinister nature whereas prolapse of the pupillary margin is of little consequence.

Conjunctival flaps, however neatly stitched, however complete, are subject to the same result—they are an absolute failure in preventing prolapse and have no *raison d'être*.

Corneal stitches would at first sight appear to be more promising, for there should be no question of their giving way, being inserted through firm inelastic structures. I have tried through-and-through corneal stitches, and they gave no

trouble, behaving perfectly well, as well as through-and-through stitches in the abdominal wall. There need be no fear about them on this account. But as a means of preventing prolapse they are impracticable. With an incision of one half of the corneal circumference I tried two stitches, one at eleven o'clock and the other at one o'clock. They were insufficient to prevent prolapse of the intact iris, or, a lesser evil, its adhesion to the wound. They have to be inserted before the lens is expressed—the tedious manipulation takes the nerve out of the patient, and he is liable to "go to pieces" on the operating table to a surprising, most objectionable degree. I came to the conclusion that not less than four stitches would be necessary to be effective and gave the procedure up.

What is the cause of iris prolapse? In my experience it does not occur until after the sclero-corneal wound has become sufficiently sealed to permit of intraocular tension being re-established. Then it is burst open when the patient gives a squeeze with the orbicularis and the iris is swept into the wound with the gush of escaping aqueous—if intact it is ballooned out by the pressure of the aqueous *from behind.*

In the days when I was expressing lenses upright, I was unable to adopt Hess's peripheral iridectomy, for they put their noses through the buttonhole and got caught up and were very difficult to manage. I was therefore compelled to adopt the ordinary complete iridectomy and got the best results with a sclero-corneal wound, taking the smallest possible nip, out of the iris, before the lens was expressed. (Iridectomy after the lens is expressed in its capsule is impracticable on account of the risk of the patient rolling his eye about and squeezing out vitreous.)

The tendency of the pupil to draw up in these cases of complete iridectomy makes an incision ending forward in the cornea inadvisable.

Since working out the method of expressing the lens in all cases of senile cataract as a tumbler—lower edge foremost (vide *Archives of Ophthalmology*, No. 3, 1926)—I have been able to use Hess's peripheral iridectomy. With the incision of 180° necessary for intracapsular extraction a single buttonhole at twelve o'clock gives fairly good results, but there is still some liability to incarceration at the angles. I succeeded in

getting over this by adding another buttonhole at each angle —i.e., three in all at ten o'clock, twelve o'clock and two o'clock—the incision being sclero-corneal. There should be no more hesitation in making three peripheral iridectomies than in making one. Eserine ointment should be put in the conjunctival sac on the operating table, and every second day subsequently for a few times.

Before leaving this matter I wish to deal with what at first sight would appear to be the most logical method of preventing prolapse—to attack its cause by depriving the patient of the power to use his orbicularis until the sclero-corneal wound was healed. Novocain has been boomed as a paralyzing agent, but in my hands it has not proved satisfactory on the operating table—with patients who would have behaved well anyhow it left nothing to be desired while with those who were determined to misbehave it was useless—in other words, by paralyzing the sensory nerves it does away with reflex contraction and reduces the muscle tone so that the lids tend to lie open, but the motor nerves are unaffected and the muscle remains under the control of the will. I have tried quinine urea hydrochloride in strengths far above those recommended and found it useless except in rare instances; where one was lucky enough to strike the trunk of the facial nerve any irritant would do this. In searching for a more positive method of control I put a stitch on a rubber button through the eyebrow and tied it to a piece of adhesive plaster on the forehead so that the upper orbicularis was trussed and immovable, but the first few cases demonstrated that this was not going to prevent prolapse—rather the reverse—presumably because it removed the normal support of the orbicularis in the upper fornix over the wound, while leaving the lower lid power to act. Thinking over the results in this last case I have come to the conclusion that paralysis of the orbicularis during the period of convalescence would not be an advantage but the reverse— in depriving the sclero-corneal wound of the support given by the normal tonic contraction of the lids we should be removing nature's splints, for which pads and bandages are no substitute. It is more than possible that many of the eversions of the corneal flap of which I have heard lately are the outcome of the partial paralysis

induced by novocain, for I do not remember hearing of them before the use of this drug became the vogue. It is not peripheral paralysis which is required, but inhibition of the sudden squeeze, for which there is no drug like bromide of potash, given in adequate dosage—80–100 grains the night before operation.

The Treatment of Established Prolapse.

If prolapse occurs it is inadvisable to interfere with it until twelve or fourteen days have elapsed after operation. If interfered with earlier a general anæsthetic is required—for, however well cocainized, these patients are always in an irritable mood, struggle in spite of their better judgment, and are exceedingly likely to burst the whole wound open unless sufficient time has elapsed for it to have healed soundly. There is nothing to be gained by interfering earlier, as from the prolapse itself there is no danger of sepsis.

The Drawn-Up Pupil.

A pupil requires to be much drawn up before it seriously interferes with vision. I have seen many a patient with a greatly drawn-up pupil able to thread a needle or to see for rifle shooting. For lowering a drawn-up pupil there are four procedures.

1. To make a small incision at twelve o'clock, pass small scissors in, get them astride the iris, and cut it. This looks very simple, but is far from being so. When the aqueous escapes the iris is pushed up against the cornea and it is very difficult—often impossible—to get a blade of the scissors in front of it. It is a proceeding I do not advise.

2. To do an iridectomy below at 6 o'clock through a sclero-corneal incision. It is for practical purposes impossible to do an iridectomy short of the wound itself, and the result is a bad cat's pupil.

3. To do an iridectomy through a transverse incision in the cornea just where the lower edge of the pupil is desired to be. I insert a sharp cataract knife at *right angles* to the surface of the cornea, where we want the lower edge of the pupil to

be and make an opening just large enough to get in a pair of forceps. I insert a fine pair of iris forceps, get hold of the margin of the iris, bring it out and cut off the smallest piece possible. This leaves a pupil which looks normal and is in normal position. The scar in the cornea is out of the way of the pupil and is so fine as to be visible only on careful inspection—if the incision has been made at right angles to the surface of the cornea. If it has been made obliquely it will be more visible in proportion to the obliquity with which it has been made.

4. To pass in a knife on the flat just behind the sclero-cornea, at five or seven o'clock, according to the eye—right or left—drive it right across in front of the iris to the opposite sclero-cornea. So far no leakage will have occurred if the knife is held lightly. Then twist the edge of the knife 30° edge back, lean the point well into the vitreous, and withdraw it, raising the handle in so doing so as to keep the point leaned into the vitreous and the back of the blade pressed against the sclero-corneal incision. If a straight cataract knife in first class order is used this way one never fails to cut the iris—the secret of success lies in using the knife to make a *draw out*. If the knife is simply pushed against the iris it will not cut however sharp it may be—the iris simply recedes before it.

Some claim great advantages for a sickle-shaped knife for this and similar purposes. The first objection to such an instrument is that no instrument maker has discovered a method of sharpening a knife with a concave edge, with anything like the perfection with which he can sharpen a straight or convex bladed knife. The next point is that such an instrument violates the principle of a draw out—one cannot be made with it. The principle in the oriental sword which is convex on edge is sound as a slash with it implies a draw out.

Lowering a drawn-up pupil by one of these last two methods is in skilled hands one of the simplest and safest of operations.

STUDIES IN INTRAOCULAR PRESSURE.

By Dr. LOUIS BOTHMAN and Dr. SEYMOUR J. COHEN, Chicago.

(From the Departments of Ophthalmology and Pharmacology, University of Illinois, College of Medicine, Chicago.)

IN the discussion on the physiology of the intraocular pressure by Hill and Starling (1) it was the consensus of opinion from experimental investigation that the intraocular pressure varies with the blood pressure within certain limits. However, as early as 1885 Howe (2) showed that the variation of intraocular pressure is a measure of venous pulse. Parsons and Snowball (3) by recording simultaneously blood pressure and intraocular pressure showed that the pressure in both systems varied in the same direction. Their conclusions were that the variations in intraocular pressure were due to the changes in the volume of the intraocular vessels. This evidence was indirect for they did not examine the fundus to study the changes in its vessels.

After considerable experimentation on intraocular pressure, Henderson and Starling (4) arrived at the same conclusions as Parsons, chiefly that the intraocular pressure is a function of the blood pressure in the intraocular blood vessels and varies directly as the latter. The same criticism applies here also, that they did not observe the fundus of the eye and their conclusions in regard to the changes in the volume of the intraocular blood vessels are derived from indirect evidence.

In a series of papers on the intraocular pressure and the tension of the eye, Elliott (5) reviews the experimental work of other investigators and among his conclusions states that the systemic blood pressure tends, as it rises and falls, to exert a corresponding influence on the intraocular pressure. Marx (6)

recorded changes in intraocular and intraspinal pressure simultaneously, but did not show their relation to blood pressure. However, his conclusions show that both systems vary in the same direction when adrenalin and pituitrin are injected intravenously but he does not show what occurs to the blood pressure.

Lastly, among the factors, mentioned by Seidel (7), which regulate intraocular pressure are: changes in general blood pressure, changes in circulation through the eye, elasticity of the bulbar wall and inflow and outflow of ocular fluid.

It is very apparent from the foregoing data that practically all the investigators are of the opinion that variation in intraocular pressure depends upon changes in the general systemic blood pressure and specifically on changes in the volume of the intraocular blood vessels, although none of them have looked directly into the fundus to observe these changes in the volume of these vessels. Their conclusions are all based on indirect evidence. Our investigation into this work was done to review the earlier work and to determine the action of drugs that alter general systemic blood pressure and in this way to study the changes in intraocular pressure, and second, by direct observation of the fundus, to follow the changes in these vessels as intraocular pressure varies.

METHOD.—The work was carried out on the eyes of dogs and albino rabbits. Three different methods were used in the study of these changes. The first series of observations were made after introducing a small spinal puncture needle into the anterior chamber of the eyes of dogs under ether anæsthesia, and connecting the needle with a water manometer. This pressure within the eye was correlated with the simultaneous blood pressure, determined by a canula in the femoral artery. The second series of observations were made after enclosing the eyeball, with and without the muscle attachments, in an air-tight chamber and connecting this oncometer with a water manometer. A simultaneous blood pressure from the femoral artery was also taken. The third study was by direct observation of the choroidal and retinal vessels in albino rabbits under chloretone anæsthesia, through a Gullstrand electric ophthalmoscope (Bausch and Lomb model) using 9, 14 and 20 magnifications.

RESULTS.—With a water manometer attached to a needle in the anterior chamber of the eye, and by using drugs which cause either a hyper- or hypotension, marked changes in the intraocular pressure were produced.

Adrenalin, pituitrin and nicotine usually produce a marked hypertension when given intravenously, while nitroglycerine produces a marked hypotension. The results of these drugs on intraocular pressure are as follows:

Adrenalin, *1cc* of 1–1000 solution injected intravenously in a dog, produces an average rise in blood pressure of *80mm* of Hg and an average rise in intraocular pressure of *25mm* of water.

Pituitrin, 2 ampules injected intravenously, produces an average rise in blood pressure of *90mm* of Hg and a rise of *30mm* of water in intraocular pressure.

Nicotine, *5mg* injected intravenously, causes an average rise in blood pressure of *60mm* of Hg and a rise of *18mm* of water in intraocular pressure.

Nitroglycerine, in *2mg* doses injected intravenously, produces an average fall in blood pressure of *40mm* of Hg and a fall of *15mm* of water in intraocular pressure.

TABLE I.

Drug.	*Dose.*	*Change in Blood Pressure.*	*Change in Intraocular Pressure.*
Adrenalin.....	*1cc* 1–1000	+*80mm* Hg	+*25mm* H_2O
Pituitrin.....	2 ampules	+*90mm* Hg	+*30mm* H_2O
Nicotine.....	*5mg*	+*60mm* Hg	+*18mm* H_2O
Nitroglycerine.....	*2mg*	−*40mm* Hg	−*15mm* H_2O

These results indicate that increasing or decreasing the blood pressure produces a simultaneous change in intraocular pressure. This change is abrupt, does not last long and follows closely the fluctuation in general systemic blood pressure. This is best shown with stimulation of the nerve. Stimulation of the central end of the vago-sympathetic in the dog produces very little change in blood pressure and practi-

cally no change in intraocular pressure. (Other investigators (3–5–9) have reported that stimulation of the central vago-sympathetic in the dog produces a rise in intraocular pressure. This is due, however, to an exophthalmos, and the protrusion of the eyeball forward, presses against the lids and needle, which causes the apparent increase in intraocular pressure. If the margins of the lids are cut away and the eyeball has nothing to push against, then stimulation of the central vago-sympathetic nerve produces practically no change in the intraocular pressure.)

Stimulation of the peripheral end produces a marked fall in blood pressure with a drop in intraocular pressure. When the stimulation is stopped, the blood pressure and intraocular pressure rapidly rise to normal. After the injection of atropine and paralysis of the vagus nerve endings, stimulation of the peripheral end of the vagus produces no change either in blood pressure or in intraocular pressure.

Compression of the common carotid artery on the same side as in our observation, producing a partial cerebral anemia, causes a fall in intraocular pressure. When the ligature is released and the blood flows back into the head the intraocular pressure rapidly returns to normal.

All these observations, with the fact that all changes occur within one-half to one minute, lead us to believe that these changes in intraocular pressure are directly dependent on changes in general systemic blood pressure. These changes may be due to one or all of four possible factors:

1. Increased secretion of aqueous.
2. Changes in size of pupil.
3. Changes in volume of eyeball.
4. Changes in size of caliber of vessels within the eye.

Changes in Secretion of Aqueous.—With the needle in the anterior chamber, attached to the water manometer, intravenous injection of 5*mg* of pilocarpine produced first a drop in blood pressure of 20*mm* of Hg with a drop of 6*mm* of water in intraocular pressure, followed immediately by a rise of blood pressure of 30*mm* of Hg add rise of 10*mm* of water in intraocular pressure; both then gradually fall to normal. The changes here, too, followed general systemic blood pressure changes. The use of pilocarpine after atro-

pine produced no demonstrable change in blood pressure or intraocular pressure.

From these results we do not believe that the rise of intraocular pressure is due to increased aqueous flow, because of the primary drop observed and because the increase was not maintained although other signs of general pilocarpine action persisted for some time.

Local Mechanical Action of the Pupil.—Variations in the size of the pupil *per se* have no effect on the intraocular pressure, for stimulation of the central end of the sympathetic nerve in the dog and rabbit produces maximum dilatation of the pupil with very slight alteration of the blood pressure, and no change in intraocular pressure. Also, application of eserine to the eye produces a small pupil but no change in the intraocular pressure (8).

Changes Due to Variation in the Size or Volume of the Eyeball.—By using an air-tight oncometer enclosing the entire eyeball, first with all muscles and vessels intact, and again with only the optic nerve intact, the volume was measured with a water manometer. By using adrenalin hydrochloride (Parke Davis and Co.), nitroglycerine, stimulation of the central and peripheral ends of both the vagi and sympathetic nerves in dogs and rabbits, no change in the volume could be demonstrated.

Changes Due to Variations in the Caliber or Volume of the Retinal or Choroidal Vessels.—By direct examination of the retinal and choroidal vessels, through the Gullstrand ophthalmoscope, in white rabbits, some very definite changes were observed.

With 1*cc* of 1–10,000 adrenalin hydrochloride injected into the ear vein of the animal a definite constriction of the choroidal and retinal arteries, and a marked dilatation of the veins could be seen. During our first observation with a monocular eyepiece, the dilatation of the veins was so marked that we were unable to see the arteries of the choroid. But on closer observation with a binocular eyepiece the arteries were distinctly seen to contract at first and then dilate, but the flushing of the veins was so marked from the beginning that the fundus appeared one mass of red. A similar phenomenon was observed with 2*mgms* of nicotine. With

2mg of nitroglycerine a marked blanching of the choroidal and retinal vessels was noted. With *3mg* of pilocarpine a blanching followed immediately by a decided flushing of the choroidal and retinal veins was observed. Injection of *10mg* of atropine caused no change in the vessels. After atropine, no change was produced by pilocarpine. During death of the animal a gradual pallor of the choroidal and retinal vessels was noted. These changes observed by the direct observation of the vessels of the fundus show quite conclusively that the changes in intraocular pressure must be due to changes in the volume of the intraocular blood vessels, and especially those of the venous system.

Summary.—1. Changes in normal intraocular pressure due to the intravenous use of drugs are dependent on variation of the systemic blood pressure. This increase in pressure causes a change in the volume of blood in the choroidal and retinal vessels as seen by direct observation.

2. Changes in the size of the pupil *per se* produce no change in intraocular pressure in the normal eye.

3. There is no demonstrable variation in the volume of the eyeball produced either by action of drugs or by nerve stimulation.

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THE PELVIC ORGANS AS FOCI OF INFECTION IN INFLAMMATORY DISEASES OF THE EYE.[1]

By Dr. WILLIAM L. BENEDICT, Section on Ophthalmology, Dr. WILLIAM H. Von LACKUM, Section on Urology, and Dr. ALLEN A. C. NICKEL, Division of Experimental Bacteriology, Mayo Clinic and The Mayo Foundation, Rochester, Minnesota.

CHRONIC infection in organs easily accessible to bacteria from extracorporeal sources gives rise to inflammatory diseases of the eye through metastasis, or the transfer of bacteria through the blood stream. As the bacteria are carried in small numbers in the blood, attempts to find them are usually unsuccessful. In a series of experiments reported by one of us in 1921 (1), it was demonstrated that iritis was produced in animals by bacteria carried to the eye by the blood stream. The localization of the lesion in the eye also indicates that the mode of transmission is by the blood stream, as the examination of specimens shows small blood vessels and capillaries plugged with thrombi containing large numbers of bacteria. The exact mode of entrance of the bacteria into the blood stream may not, in all instances, be the same, but that they are carried by phagocytes may be a sufficient explanation for their mode of travel.

Chronic infections that cause metastatic diseases in the eye do not often produce local abscesses, although small pockets of pus may sometimes be found around roots of teeth and in the deeper crypts of the tonsils. Infections that become more active and cause wide destruction of tissue with the formation of abscesses bring about systemic reaction.

[1] Read before the American Ophthalmological Society, Hot Springs, Virginia, June 14, 1926.

Only in rare instances do such foci cause metastatic disease of the eye. On the contrary, a focus of infection that shows little or no external evidence of disease not infrequently harbors organisms that give rise to serious diseases of the eye. Such infections do not spread by direct extension beyond a small area and practically never beyond the organ in which they are situated without causing a systemic reaction.

There is convincing clinical and experimental evidence that foci of infection around devitalized teeth and in diseased tonsils give rise to disease of the uveal tract. Infection in the prostrate also may be the source of many cases of uveitis. That the same organism may be present in periapical lesions, tonsils and prostrate has been shown by bacteriologic investigation, and that too in a person without symptoms of disease associated with any of these organs. Yet such bacteria were believed to be responsible for metastatic inflammations in the eye.

It is difficult to explain the intermittent character of acute inflammation of the eye except on the basis of focal infection. Attacks of iritis, separated by months or even years, may be due to a single focus of chronic infection, such as chronic prostatitis, yet the eye may remain free from symptoms during the intervals between attacks. As there is no periodicity of physiologic function of the teeth, tonsils and prostate, it is not likely that the intervals between attacks can be explained on any basis except systemic reaction. It has been noted, however, that following the extraction of an infected tooth, enucleation of a diseased tonsil, or massage of a diseased prostate that focal reaction (recurrence of the inflammation of the eye) appears. This can be explained on the basis of the transfer of a large number of bacteria or their toxins through the blood stream, access to which has been made more available by means of rupture or increased permeability of vascular walls. The reaction in the eye occurs in from twelve to thirty-six hours, and may be more severe than attacks suffered previously. We have no clear evidence as yet that toxins of bacteria, without the presence of the bacteria, cause inflammatory lesions of the eye in a natural manner. Hence it becomes necessary to search for and, if

possible, remove the bacteria from a focus of infection, besides carrying out reasonable hygienic measures.

In cases of scleritis and sclerosing keratitis we have examples of chronic inflammatory disease of the eye which in women is often periodic and progressive. The character of the disease is typical of metastatic focal infection; that is, it is not suppurative, it is intermittent and painful, and responds to treatment by the removal of foci of infection and the use of autogenous vaccines. The periodic exacerbations occur quite regularly with the function of menstruation, beginning a day or two before the flow and lasting from two to five days. The remission occurs between menstrual periods. Women who have had scleritis with exacerbation coincident with menstruation have been free from attacks during pregnancy and lactation, only to have the disease resume its periodic character with the reëstablishment of regular menstruation. This indicates a relationship between the focus of infection and the eye that has still a further determining factor than the balance between the bacteria and resistance of the body, that is, the function of the organ containing the focus. Even here, however, it is not the physiologic functions of the organ that bring about the change, but the ready access of bacteria to the blood stream about the cervix as the uterus becomes congested. With the history and clinical evidence of this relationship between the cervix uteri and the eye established, bacteriologic examinations were made in a few cases, and local treatment to the cervix, together with vaccines, instituted.

ILLUSTRATIVE CASES.

CASE 1.—A woman, aged thirty-one, came to the Mayo Clinic in the summer of 1921 because she had had severe inflammation of the right eye for three months; it had appeared suddenly, and in a few days the eye became very painful and congested. Her left eye had been amblyopic since infancy and was divergent. Previous to the onset of the inflammation her right eye had been normal, with vision $\frac{6}{6}$. Under treatment the inflammation subsided and the eye was quite clear but the inflammation returned at the time of the next menstrual period, again subsided after a few days but did not disappear. After the third period the inflammation decreased very little.

Examination in the clinic revealed typical episcleritis of the right eye with sclerosing keratitis. A von Pirquet tuberculin test was negative. The teeth showed no evidence of infection. The tonsils had been removed three years previously. A Wassermann reaction was negative. Under local treatment the eye gradually improved for a few days but again became markedly inflamed with the onset of the next menstruation. When menstruation had subsided pelvic examination was repeated and reported to be negative. Cultures made from the secretion around the cervix, however, revealed a hemolyzing streptococcus. Vaccine prepared from this culture was then given in addition to local treatment to the cervix. Local and focal reaction followed the first few administrations of vaccine but the eye cleared within a few weeks. There have been no recurrences.

CASE 2.—A woman, aged sixty-one, was referred by an oculist who had treated her for eight months for periodic scleritis of the right eye without satisfactory results. There was no clinical sign of tuberculosis; the Wassermann reaction was negative; there was one devitalized tooth and a slight amount of pyorrhea, and a small rectocele and cystocele were present with slight discharge of mucus. A culture of hemolytic streptococci was grown from the secretion around the cervix and a vaccine made from it. Atropin and epinephrin solutions were applied to the eye, and vaginal douches of an antiseptic solution were prescribed. The inflammation subsided after the vaccine treatment and a year later had not returned.

As has been previously stated, chronic prostatitis has been shown to cause inflammatory changes in the eye. Prostatitis, either of hematogenous or urethral origin, may be entirely asymptomatic or may produce one or all of three groups of symptoms: urinary, sexual, or general. In a definite percentage of cases the organs of special sense, particularly the eye, exhibit general metastatic symptoms or lesions.

Prostatic infections produce metastatic lesions in one of three ways: by toxin, reflexly, or by infection. The lesions of the eye are distinctly caused by infection. In 25 per cent of the cases of prostatitis the prostate is normal on digital examination. In 33 per cent the urine is normal, and in many there are no urinary or sexual symptoms. Thus, it is important to investigate the prostate, irrespective of local signs or symptoms.

By far the most accurate means of diagnosing prostatic infection is microscopic examination of secretion; unfortunately it is not always possible to express secretions for this purpose. The presence of an unusual number of pus cells means infection. The finding of small clumps of pus cells or a few large degenerated leukocytes, is likewise of diagnostic value. In the latter cases recognized as latent infections, it is necessary, as a rule, to subject the patient to provocative treatments, consisting of massage of the prostate, wide posterior urethral dilatations, and often repeated injections of solutions of silver nitrate in order to obtain secretions of diagnostic significance.

Bacterial prostatitis (prostatic secretions that contain bacteria without pus cells) seems a definite entity and represents a rarer group of infections. This can only be diagnosed by cultures of the prostatic secretions. Several methods have been used to obtain such secretions uncontaminated, but none has proved uniformly satisfactory.

The method employed in the Mayo Clinic consists of irrigation of the bladder and urethra and cleansing of the glans with sterile sodium chlorid solution. Following this preliminary care, the secretions are expressed and collected in a test tube of 1 per cent gelatin-Locke solution in which container they are sent to the bacteriologist. Secretions from seminal vesicles are expressed and collected after the same preliminary procedures, and if it seems desirable to collect secretions separately from prostate and vesicles, irrigation between expressions is necessary.

The treatment, as well as the diagnosis is necessarily a urologic procedure; the former consists primarily of the removal of other primary foci, and secondarily of local treatment.

It is relatively difficult to produce lesions of the eyes of rabbits experimentally, especially by the intravenous route unless the conditions are suitable. In promising cases, however, lesions of the eye, consisting chiefly of those due to various injections, scleral hemorrhages and iritis, have been produced experimentally with strains freshly isolated from the teeth and tonsils. More recently with similar technic, we have been able to produce lesions of the eye with cultures from the prostate and cervix. If the cervix is the suspected

focus, the method of obtaining a culture is as follows: A sterile speculum is inserted. The first swab wipes off the material covering the external os and this is used as a control, the second swab is inserted directly into the cervical canal or ruptured cervical cysts and the material obtained is used for a culture. If the prostate is the suspected focus, the procedure is similar to that described elsewhere in this paper. All cultures are then made as a routine on aërobic blood-agar plates, in tall tubes of glucose brain broth and in soft glucose brain agar according to Rosenow's method. The blood-agar plate serves to control contamination, and the glucose brain broth and agar furnish a gradient of oxygen tension. The latter also preserves the primary culture for future use and indicates by the number of colonies, the degree of infection present in the focus.

The organism usually isolated is a hemolyzing streptococcus, less commonly a green-producing streptococcus, and in a few cultures a Gram-positive bacillus was evidently also of etiologic significance. Many of the streptococcus strains when freshly isolated, did not grow on aërobic mediums, and approximately 44 per cent of the cultures, especially cultures from the prostate did not grow when plated directly on a blood-agar plate but did grow in glucose brain agar and glucose brain broth, which shows the necessity of having "gradient of oxygen tension" for culturing these organisms. On blood-agar plates the hemolyzing streptococcus colonies are a trifle larger than typical hemolytic streptococcus colonies and there is a narrow zone of almost complete hemolysis immediately adjacent to the colony, but this zone is not as wide as that of a typical hemolytic streptococcus. In a liquid medium the organism often occurs in pairs and is similar in structure to the *Streptococcus viridans;* in other instances it occurs in typical hemolytic streptococcus chains. When plated on blood agar, after having been subcultured a number of times in glucose-brain broth, this hemolyzing streptococcus frequently grows a typical *Streptococcus viridans.*

From 2 to 12*cc* of the glucose-brain-broth culture is injected intravenously into rabbits, depending on the size of the rabbit and the nature of the case. If the first injection has no effect on the rabbit, the injection is repeated once or

twice with subcultures on successive days. If a rabbit does not develop a lesion of the eye in seven days after the last injection, the result is considered negative. In all these animal experiments the lesion produced in the eye is directly dependent on the elective localizing power of the organism injected (Tabulation). Therefore, it is essential that freshly isolated cultures be injected as soon as they have grown in glucose brain broth, preferably by the time they are eighteen hours old. Cultures that have been allowed to stand around for days lose their localizing power and when injected produce no definite lesions.

Four out of six strains from the cervix, and ten out of eighteen strains from the prostate from patients with lesions of the eye, produced macroscopic lesions in the eyes of rabbits. Six cultures of the cervixes of six patients who had lesions of the eye were injected into sixteen rabbits, and five (31 per cent) developed macroscopic lesions of the eye. Similarly, eighteen cultures from the prostate of eighteen patients who had lesions of the eye were injected into forty-five rabbits, and thirteen rabbits (29 per cent) developed macroscopic lesions of the eye. Thus, of a total of sixty-one rabbits injected with these twenty-four strains, eighteen (30 per cent) developed lesions of the eye in contrast to 7 per cent of 181 animals injected with seventy-nine strains obtained from teeth, tonsils, prostate and cervixes of patients whose conditions were not definitely diagnosed. In another series of experiments with cultures from teeth, tonsils, prostate or cervix, 335 strains from patients having lesions other than those of the eye were injected into 798 animals, and less than 1 per cent developed lesions of the eye. With the strains from the prostate and cervix of patients with lesions of the eye, 30 per cent of the animals injected developed lesions of the eye. Only 3 per cent developed lesions of the joints and kidneys, and 2 per cent lesions of the heart, muscle, stomach or gall-bladder. A few such aberrant localizations occur in any series of animal injections and they parallel quite well similar localizations in the control series. Thus, in the joint they are 3 and 7 per cent, respectively; in the heart, stomach and gall-bladder, 2 and 3 per cent; in the muscle, 2 and 5 per cent; in the kidney, 3 and 5 per cent, and in the liver

0 and 2 per cent; in neither of the latter two was there localization in the skin (Tabulation). In many cases the condition of the eye was also chronic; in some the focus was questionable, and in some there had been no recent exacerbations. Excluding such cases we have made eight cultures from the cervix or prostate for experimental purposes and injected them into eighteen rabbits. Lesions of the eye were produced in twelve rabbits, 67 per cent of the animals injected.

CONCLUSIONS.

1. The prostate and the cervix may be potential foci of infection in the production of lesions of the eye.

2. The causative organism isolated from these foci is usually a slightly hemolyzing or green-producing streptococcus requiring partial tension methods for its isolation.

3. Local treatment and autogenous vaccines made from strains obtained from experimental lesions of the eye in rabbits often greatly benefit the patient.

TABULATION.

LOCALIZATION IN ANIMALS OF BACTERIA ISOLATED FROM THE PROSTATE AND CERVIX OF PATIENTS WITH LESIONS OF THE EYE.

SOURCE OF CULTURE	*Cases studied*	*Animals injected*	*Cases positive in animals*	PERCENTAGE OF ANIMALS WITH LESIONS OF THE								
				Eye	*Joint*	*Heart*	*Muscle*	*Skin*	*Stomach or duodenum*	*Kidney*	*Gall-bladder*	*Liver*
Prostate.....	18	45	10	29								
Cervix.....	6	16	4	31								
Total.....	24	61	14	29.5	3	2	2		2	3	2	
Controls.....	79	181		7	7	3	5		3	5	3	2

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OBSERVATIONS ON GLAUCOMA IN MORGAGNIAN CATARACT.[1]

By Dr. ARNOLD KNAPP, New York.

A CATARACT which in its hypermaturity has reached the Morgagnian stage is apt to develop attacks of congestive glaucoma. The cause for the glaucoma has been variously explained and the treatment is not always easy or satisfactory.

Harold Gifford ("Glaucoma from the Absorption of Senile Cataract," *A. J. O.*, 1918, p. 83) believes that the degenerating cortex causes toxic products which are absorbed and produce the glaucoma. Feingold (in the discussion following Gifford's paper) thinks that the irritation of the ciliary body by the subluxated (shrunken) lens seems to be the only plausible explanation for the glaucoma. This Gifford does not accept, believing that the toxicity of the lens cortex is the bigger factor, rejecting the theory as to traction upon or irritation of the ciliary body.

Recently two cases of this character have come under my observation which illustrate some of the points in question:

CASE REPORTS.

1. *S. R.*, 57 years old; right cataract extraction in capsule, 1913. Ten years later, August, 1923, patient returned with the statement that the left eye which now presented a Morgagnian cataract, had been inflamed for two weeks. The cornea was hazy; T. 55. Pilocarpin and general treatment improved the condition, and on October 27th, com-

[1] Read before American Ophthalmological Society, Hot Springs, Va., June, 1926.

bined extraction was performed. It was difficult to grasp the capsule with the Kalt forceps; it eventually tore and the nucleus was expressed. There was very little fluid cortex present. The pupil was black after the coloboma was made, and the nucleus had undoubtedly sunken back of the iris. In convalescence the anterior chamber remained shallow, T. —. Choroidal detachment. December 1st: cornea dull, anterior chamber deep; posterior synechiæ; T. 45. December 18th: Lagrange operation performed dividing capsular adhesion above. Small vitreous prolapse abscissed: slow recovery. January 7, 1924: S +12 $\frac{13}{200}$, Tn, updrawn pupil, adherent capsule. April 14th: irido-cyclitis, deep vessels and opacity in cornea below. Pupil adherent to thickened capsule attached to wound. T. —. July 22d: T. 44. April 14, 1926: T. 26; iridotomy advised.

2. *H. M.*, 76 years old, had had a cataract extracted from his left eye in 1910. The cataract in the right eye gradually became hypermature. On January 7, 1925, the right eye became red and painful, ciliary congestion and tension of 42. Gradually with miotics and general treatment the eye became white, tension 26. On April 27th another attack with ciliary congestion, single posterior corneal deposit, a few slight synechiæ. T. 38. June 1st: the eye was white and T. 22. Iridectomy was then performed without incident and tension was reduced to 18. The patient was not seen for three months. On October 1st, there had been no inflammatory symptoms but the tension was found to be 38. In the coloboma the nucleus could be easily seen in the capsular bag, the cortex was clear, a clean gap above showed a shrunken capsule, iris tremulous. With miotics the tension was brought down to 31 where it remained, and on November 5th, the cataract was extracted. The lens was readily subluxated with the blunt capsule forceps and extracted in the capsule in the usual way. The recovery was prompt and uneventful. A few adhesions formed along the lower pupillary margin. No return of tension. At a recent examination six months later, the sight with correcting glass was $\frac{3}{10}$. Field contracted; optic nerve atrophic; tension normal.

The attacks are suggestive of a low grade uveitis in which the increased tension is the most striking feature. In Case 2, after the iridectomy tension returned without any inflammatory symptoms. Elimination of the usual toxic factors were

unavailing, so that the local condition in the Morgagnian cataract seemed to be the cause for the return of tension. If the absorption of the cortex is the toxic agent as regarded by Gifford, why, in these two cases, did not symptoms develop before the cortex was absorbed, and if the liquified cortex be phlogogenic why do we not see more frequently any reaction on the part of the iris and ciliary body, and why is the glaucoma always the important complication?

It frequently happens, in opening the capsule in hypermature cataracts, that the milky fluid gushes forth into the anterior chamber. Elschnig (*Graefe-Saemisch*, 2d. ed., 1922, p. 1190) says that this milky fluid has a pronouncedly irritating action on the eye and that the aqueous should be repeatedly evacuated during the operation. In my experience this irritation has not been noticeable, and in fact it has always seemed remarkable that this milky fluid is so well born by the eye. Furthermore, if the fluid cortex were toxic why should it not at least cause a few synechiæ? I cannot therefore subscribe to Gifford's belief and feel that the displaced nucleus must cause some mechanical irritation of the ciliary processes and thus produce the glaucoma.

The method of treatment in these cases is undoubtedly extraction of the cataract in the capsule, but the technique is not easy. Elschnig (l. c. p. 1190) says that in Morgagnian cataract, the lens can be easily expressed by Smith's technique, though it can also be expressed with capsule forceps and iridectomy. Elschnig however acknowledges that the lens capsule is readily torn by the forceps. This facility of extraction in the capsule does not agree with Smith's experience, and a consideration of the mechanical factors present in this type of hypermature cataract proves that it is always a difficult form to express in the capsule by the Smith technique (Smith, *The Treatment of Cataract*, p. 9, Calcutta, 1910), and vitreous is apt to be lost. In attempting the extraction in the capsule with preliminary dislocation with the blunt capsule forceps, the forceps takes a firm hold with difficulty on account of the fluid cortex, as if the capsule were slippery, and when it is finally grasped it ruptures easily. If the capsule is partly subluxated as in the second case, the extraction is easier.

In the first case, the capsule ruptured during the forceps manœuvre a capsulotomy operation was done, which was followed by irido-cyclitis and return of tension. This required another operation and now the pupil is updrawn and closed; an iridotomy is now necessary. In the second case, the forceps succeeded in dislocating the cataract, and the cataract was extracted in its capsule. This permanently relieved the increased tension.

In general, the treatment of this condition is of course preventive; it is not wise to allow a cataract in which the cortex is becoming fluid to advance without an operation; if attacks of glaucoma have set in, they should first be treated medically, and the cataract is then to be extracted, preferably in the capsule, when the eye is quiet; otherwise inflammatory relapses and secondary glaucoma may result.

SLIT-LAMP STUDY OF POSTERIOR LENTICONUS.[1]

By Dr. ELIAS J. MARSH, Paterson, N. J.

SINCE posterior lenticonus was first recognized and reported by Meyer, in 1888, some thirty-eight cases have appeared in the literature under this name. Careful studies of this condition, with review of published cases and full bibliography have been made in recent years by several writers, notably Mme. Gourfein-Welt, V. Jano, and Colombo, all, however, before the day of the slit-lamp. For the sake of brevity, therefore, reference will be made to this older work at this time only for certain basic observations or theories important in connection with the slit-lamp findings as reported in the few cases so studied.

Of the earlier cases a considerable number must be regarded with suspicion as not being true lenticonus, owing to mistakes either in diagnosis or in classification. In the most recent general survey of the subject, Colombo, writing in 1922, rejects all but ten of twenty-nine cases studied. He is, however, exceptionally exacting in his standard, defining true lenticonus as a congenital, transparent, hemispherical prominence, circular in outline, superposed on the posterior pole of an otherwise normal lens. Others, though less strict, are critical of many of the cases, especially the earlier ones. Von Jano, and after him Riedl, both of Elschnig's Clinic, divide the lenticonus cases into two classes, the first a hemisphere at the pole, marked off by a sharp line from the normal curve; the other a more or less truncated cone replacing the normal curvature in the middle part of the lens: for the former of these,

[1] Case presented before the Section on Ophthalmology, N. Y. Academy of Medicine, November 17, 1926.

Elschnig's term "*lentiglobus*" is advocated. The cases of general deformity of the posterior part of the lens in buphthalmic eyes, reported or collected by Gourfein-Welt are hardly to be considered in this classification. Undoubtedly some of the reported cases belong in the category of "false lenticonus" (Demicheri) or change in the embryonal nucleus. The case reported by Mackenzie, with history of gradual onset in a middle-aged physician, "riders," fluid vitreous, and increasing opacity of the entire lens certainly suggests such an explanation.

The diagnostic signs given by the earlier writers were the luminous disc in the center of the lens (the so-called "oil-drop" phenomenon), surrounded by a shifting dark line, as seen by the retinoscope, and the greatly increased myopia of the central part as compared with the periphery. In 1892 Gullstrand published a study of the movement of the Purkinje images of the posterior lens surface, by which he was able to work out very accurately the form and the changes in curvature, and to prove that in his case the normal lens concavity and that of the cone were joined by a short convex curve, instead of a sharp angle. Thus he added another sign, of great value in differentiation from false lenticonus, viz.: the movement of the reflections on the posterior surface.

A great majority of the reported cases have more or less opacity, either limited to the cone or involving the neighboring parts of the lens, and a large number have visible hyaloid remains. In many cases it is stated that the nucleus is displaced backward toward the protrusion. These observations have had considerable influence on the discussion of the etiology of the condition. Three theories have been advanced to account for its origin, viz.: (1) increase in volume of the lens, in the nature either of a swelling of the lens fibres, or of a neoplastic growth of lens tissue; (2) hernia of the cortex through a gap in the capsule, or the yielding of a weak spot in the capsule; (3) traction at the pole during fœtal life by the hyaloid vessels. Almost all of the cases are considered to be either congenital or to have developed very early in life, though three have been attributed to trauma.

The first of these theories has gained little favor, as there is no evidence in its support and no reason given for such swell-

ing or growth, especially in young persons with clear lenses. The second is much better supported, both by argument and by observation. Fisher reports having watched the gradual development of a protrusion at the posterior pole of the lens, due, as he supposed, to a rupture of the capsule at this point in consequence of a blow on the head, the protrusion and eventually the entire lens gradually becoming opaque. Collins, on the other hand, points out that there is no reason to expect a rupture or stretching of the capsule at the pole, where it is normally thickest, but advances, as by far the most probable explanation of congenital lenticonus posterior, a protrusion of cortex through a gap left in the capsule by failure of embryonal closure, a process which must be completed at a comparatively early stage. The third theory, hyaloid traction, is supported by the large percentage of cases in which hyaloid remains or posterior capsular cataracts are observed, and is accepted by various writers, especially Colombo and Gourfein-Welt. The latter, incidentally, points out that no clinically observed case has so far come to section, while none of those anatomically studied had been recognized during life. This, it is believed, remains true.

This summary brings us to the advent of the slit-lamp. A careful search of the recent literature has brought forth four cases studied and reported with the aid of this instrument. The first, naturally and properly enough, is by Vogt, the second by Riedl, who also observed with the slit-lamp the case previously reported by v. Jano, while the fourth was presented by Whiting to the Royal Society of Medicine. Beside these, Clemesha and Blaauw of Buffalo reported a case under this title, studied with the slit-lamp, but Dr. Blaauw has since said (*P. C.*) that he now considers this case to be of nuclear origin, rather than a true lenticonus.

Vogt remarks that one might have expected lenticonus to be one of the conditions in which the slit-lamp would be most helpful, the narrow slit beam enabling us practically to palpate the distension and to measure exactly its form and dimensions, giving us an anatomical section free from the distortion caused by hardening and cutting. He then describes the findings in a 31-year-old female patient, whose left eye is normal, and the right also aside from the special deformity

under consideration. At the posterior pole of the right lens is a cup-like depression, measuring in depth about one-third the sagittal diameter of the lens proper, which is about the same as that of the other eye. The circular margin of this disc, when illuminated by the slit-beam, becomes a brilliant ring of fire, which Vogt calls the ring reflex, and considers to be a new diagnostic sign of the first importance, especially in distinction from "false" lenticonus. Study of the lens capsule shows a "fairly sudden" bending away from the normal curve to that of a flattened cone. There is some opacity of the outermost layers, involving in places even the senile nuclear layer, which follows the abnormal line of curvature. The embryonal nucleus, on the other hand, is entirely normal, a condition which Vogt considers to speak for a late fœtal or early post-natal origin of the condition. Great importance attaches in his opinion, to the position of the hyaloid remains, which are nasal from the pole (a position which he has elsewhere shown to be their normal one) just touching the edge of the protuberance. This, he considers, if it does not definitely dispose of the hyaloid traction theory, at least proves that it does not apply to all cases. On the capsular hernia theory the present findings afford insufficient bases for an opinion.

Riedl's case shows a conical baying at the posterior pole, in depth about one-third of the lens diameter, and displaying Vogt's ring-reflex at the edge, a phenomenon whose brilliant and striking character impresses Riedl as strongly as it had done Vogt. The nucleus is normal in size, character, and position. The hindermost layers of the conus show a net-like clouding. No visible hyaloid remains. Fundus normal. Peripheral refraction low hyperopia, central high myopia. Other eye presents a dense posterior polar opacity.

Riedl also examined by the slit-lamp the case earlier reported by v. Jano. Here also the embryonal nucleus was normal, the posterior cortical layer thickened but fairly transparent even in the conus, through which the fundus could be seen.

The report of Whiting's case so far published in the proceedings of the section on ophthalmology is so brief as to be hardly more than a record of the fact. It is stated that the

deformity of the posterior part of the lens was well made out with the slit-lamp. The alteration of the lens contour was well shown; "it has almost a nipple on its posterior surface."

Beside these Vogt has published a slit-lamp study of a case ophthalmoscopically and retinoscopically indistinguishable from lenticonus, which is shown to be a beginning diffuse cataract of the embryonal nucleus, not recognizable as such by the older methods. He considers the term "false lenticonus," first used by Demicheri, to be rendered by the slit-lamp as indefensible as the mistake in diagnosis has been made inexcusable, preferring von Szily's "double focus lens." Riedl uses the term "central lens myopia" in this condition. A very similar case is reported by Kyrielies in an eighteen-year-old patient, due to a very faint clouding of the nucleus ("rudimentary cataracta fusiformis"). He believes that most of the reported cases of lenticonus posterior, if examined by the slit-lamp, would be found to be of this nature, true cases being, in his opinion, extremely rare.

Case Report.

Miss R. F. was first seen by me in the Paterson Eye and Ear Infirmary in June, 1914, aged nine years, coming on account of poor right vision discovered by the school medical inspector, but previously unsuspected by her. Examination at that time:

Under cycloplegia, R. V. $= \frac{15}{200} : +3.50^{s} = \frac{20}{60}$. L. V. $= \frac{20}{30} - : +1^{s} = \frac{20}{15}$. Retinoscopy of the right eye revealed the classical "oil drop" appearance, a red transparent disc at the posterior pole surrounded by a dark circle changing with the direction of the light. The retinal vessels, highly minified, were plainly visible through the "drop."

After two or three visits to the clinic the patient disappeared and was clinically lost, till she came to my office in May, 1925, complaining of asthenopic symptoms after study. *History:* had suffered a cut over right eyebrow at age of four by striking forehead against the curbstone. Had not suspected any visual difficulty until examined in school, as related above. Personal and family history otherwise negative. R. V. $= \frac{20}{200} : +3^{s} = \frac{20}{100}$. L. V. $= \frac{20}{20} - : +50^{c}$ ax. $120^{o} = \frac{20}{15}$.

Examination: A superficial, non-adherent, linear scar over the right eyebrow; otherwise lids, movements and convergence, anterior segments and pupillary reactions normal.

Under cycloplegia, left eye normal throughout, except a slight astigmatism. Right eye; retinoscopically, the oil drop appearance as already described; refraction peripherally $+ .50^{s} + 1.50^{c}$ ax. 45°, correction of which did not materially affect vision. The pinhole disc was tried without effect. Refraction through the center, about 25 D of myopia. *Ophthalmoscopically*, at the posterior pole of the otherwise normal lens appears a small red disc, perfectly transparent except that a minute opacity can just be made out at its center by careful focussing, the edge having the appearance of a dark ring or of a kryptok segment, according to the direction of the light. The posterior surface of the droplet, which is readily transparent, seems a trifle roughened or rippled. Fundus normal.

Slit-lamp finding: The lens is normal in all respects anterior to the posterior surface. The nucleus is normal in size, shape, position, and transparency, the anterior and posterior Y's readily visible. The posterior surface is normal in character and curvature outside the area under consideration. Hyaloid remnants normal in appearance and position (*i.e.*, nasal from the pole). At the point which should be the posterior pole is a fine white feather-like stellate opacity, in otherwise clear lens substance: around this as a center the posterior surface is pushed backward to form a hemisphere whose equator, at the level of the posterior capsule, is a perfect circle, with a diameter about one-fourth the equatorial diameter of the lens. The junction of the normal and abnormal curves is a sharp, practically rectangular edge, its appearance varying, with the position of the light beam, from invisibility through, a faint gray line, to a brilliant ring of fire, the *ring-reflex* of Vogt. The appearance of the ring is uniform, the whole lighting up at once, in contrast with the shifting effect given by the retinoscope. The lens substance within the hemisphere is clear cortex, the feather-like opacity at the center not approaching the surface at any point. The shagreen of the posterior reflecting surface can be followed evenly over the whole: the surface is unevenly thickened in some places, especially on the inner (lenticular) face of the capsule, but not affecting transparency. Below and nasally from the pole of the globe appears a small circular marking, with other lines within, possibly part of the hyaloid remains, but the optical distortion makes identification difficult. The post-lental space and limiting membrane of the vitreous follow closely the curve of the globe. The vitreous structure appears normal.

If the narrow slit-beam is moved from the normal posterior surface onto the globe, as it crosses the edge there

appears in addition a horizontal band of light stretching from side to side across the globe: as the beam is swung farther, this band splits into two, which, opening with a scissors motion pivoted in the center, gradually separate, to unite in the upright position as the beam crosses the middle, reversing the process on the farther side. The bands always retain a semi-circular form. Neither Vogt nor Riedl mentions this phenomenon, perhaps on account of the roughening and opacity in their cases.

If the cylindrical or "dot" beam be used, it is possible, by focussing below the globus, through normal lens, to obtain a retro-illumination which again brings out the transparent and structureless character of the globus edge. Again, the sharpness of this edge may be demonstrated by focussing this beam directly on it, when a prismatic effect is obtained, the spectral colors being cleanly split off at this point.[1] And if the dot beam is focussed directly at the pole of the globus, the entire surface is lighted up at once, with the effect of an illuminated dome, again giving the proof of a perfect hemisphere. Neither Vogt nor Riedl refers to the use of the dot beam.

COMMENT.

The slit-lamp has been of value in several ways in our understanding of this condition, as revealed by the reports of these several cases. First, the clinical as well as the anatomical distinction between true and "false" lenticonus is made final and absolute. Although this was well understood before, there is now no excuse for confusion, or even, as Vogt says, for the term "false lenticonus." Then it seems to fix definitely the division into the two groups of lenticonus and *lentiglobus*, Vogt's case and Riedl's first belonging to the former, my case and probably Riedl's second (v. Jano's) belonging to the latter. The report of Whiting's case so far available is too brief to justify deductions of any sort from it. As to etiology, any conclusions so far are negative rather than positive in character. Vogt considers, as already mentioned, that his observations on the site of the hyaloid attachment have seriously weakened, if not overthrown, the hyaloid traction

[1] It is possible that the prismatic effect does not originate here, but in the illuminating lens of the apparatus, which is not achromatic. But the prismatic colors, wherever produced, are sharply split off, at this point.

theory. My finding in this respect agrees with his: Riedl in one case reports no visible remains, and in the other does not mention them, which argues, so far as it goes, rather against the theory than for it. As to the hernia theory, Vogt admits that he has no evidence for or against. A backward dislocation of the nucleus used to be considered an argument in favor of this theory: in all four of the slit-lamp reports, the nucleus is normal in form and position. In my case the normal appearance of the posterior reflecting surface over the whole hemisphere would seem to disprove any gap in the capsule. The hemispherical form would be expected in the protrusion of semifluid cortex through a confining ring, but any such ring is apparently wanting, the margin being apparently (optically) structureless. On the other hand, the less regular form, the flattened cone with rounded angles, might be expected from the yielding of a weakened capsule over the central area. What part, if any, the blow on the forehead played in my case must remain problematical, but the whole appearance of the lens and especially of the lens substance within the cortex, together with the slight character of the cutaneous scar, make such part seem highly unlikely. It would indeed be interesting to examine some of the reported traumatic cases with the slit-lamp.

May it not be possible that the two forms of this anomaly mentioned are in fact two different anomalies, occurring at the same site, and with certain resemblances, but differing in etiology as well as in form and structure? Observations are far too few to justify an answer. It is to be hoped that as the condition becomes better known more cases may be recognized and studied by modern methods, and that cases clinically observed may in time find their way to the laboratory table.

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CORNEOCONJUNCTIVAL SUTURE FOR CATARACT OPERATIONS.

By Dr. F. H. VERHOEFF, Boston.

From the Massachusetts Eye and Ear Infirmary.

(*With two illustrations in the text.*)

RECENTLY I have devised a method for suturing the wounds in cataract operations which has proved distinctly superior to the procedure I previously employed for many years.[1] By the new method the lips of the wound are held together more firmly and there is no tendency for them to override. Its superiority is manifested especially in cases of loss of vitreous humor. In such cases any tendency for the vitreous humor to bulge open the wound is overcome as soon as the suture is tied, and, at the first dressing, twenty-four hours after operation, the wound is found everywhere firmly closed. The method is briefly as follows:

To prevent contamination of the suture the field of operation is prepared by painting the exposed skin of the eyelids, forehead, cheek, and nose with a 2% tincture of iodin, and placing a gauze mask over the nose and mouth of the patient. A fine black silk thread armed with two small half-curved needles is provided. The eye is fixed by grasping the superior rectus tendon with forceps. At a distance of 1.5 to *2mm* from the middle of the upper limbus a secure bite, about 1¼*mm* long, is taken horizontally in the cornea with one of the needles, and half of the thread drawn through. Immediately above this, by means of scissors, the conjunctiva is freed exactly at its corneal margin for a distance of about

[1] Verhoeff, F. H.: *Arch. Ophth.*, No. 5, 1916.

7mm, and then, if necessary, undermined slightly to permit it to be pulled down over the suture holes in the cornea. The two needles are now passed through the conjunctival flap from underneath, about *1¼mm* apart and at a distance of about *2mm* from its cut edge. The two threads are pulled taut, and then, by means of a strabismus hook, drawn out again in the form of two loops. These are placed to one side so as to allow the passage of a cataract knife (Fig. 1).

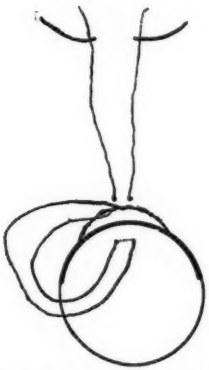


FIG. 1.—Suture ready to be tied, corneal incision made.

The cataract incision is now made as close as possible to the limbus without anywhere cutting the conjunctiva. Iridectomy is performed if desired. The free ends of the suture are now placed on the cheek ready to be grasped. The cataract is expressed and the wound immediately closed by pulling the threads downward and tying them. If irrigation is needed, this may be accomplished by inserting the irrigator tip in one angle of the wound, and opening the other angle with a spatula. If the patient is a "good actor," the irrigation may be carried out before the suture is tied.

The suture may be removed in about five days. A 20% solution of cocain is applied for thirty seconds to the flap by means of a cotton-tipped toothpick, the knot cut through, the flap pushed back, and the suture removed from the cornea with forceps.

The method is obviously simple in execution, but the

mechanical principles involved are somewhat complex, and I shall not discuss them here at length, since the advantages of the method become apparent as soon as it is actually tried on a patient. I may point out, however, that of great importance is the fact that the corneal insertion of the suture is relatively at a considerable distance from the cataract incision. It is this that prevents lateral displacement of the corneal flap and overriding of the lips of the wound, and causes the conjunctival flap to spread over and close firmly a large extent of the wound. It will be noticed, as shown in Fig. 2, that when the flap is pulled down the base becomes

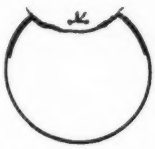


FIG. 2.—Suture tied, conjunctival flap in place.

wider and covers a larger portion of the wound than otherwise would be the case. This results from the fact that the conjunctiva folds over here.

The method in some respects is similar to that of Frisch,[1] but is superior to the latter procedure since it gives much firmer closure of the wound, insures that the suture holes in the cornea are completely covered by the conjunctival flap, and, with the raw under surface of the flap, gives an added protection against infection of the wound. Its superiority over purely corneal sutures, such as that of Kalt, is obvious. Compared to the methods which require the cataract incision to be made beneath a conjunctival flap, this procedure not only gives much firmer and more precise closure of the wound, but it has also other advantages. In the first place, it requires less skill, for it is difficult to carry an incision everywhere beneath a conjunctival flap. In the second place, in the method described, as the incision is entirely corneal, there is no hemorrhage from the wound to add to the difficulties of the operation. In the third place, there is less

[1] Frisch: *Am. J. Ophth.*, February, 1922, p. 81.

danger of an ingrowth of epithelium into the anterior chamber. I am aware that it is often assumed that this danger is greatest after a corneal incision, but in all the eyes that I have examined microscopically in which such ingrowth has occurred the incision was in the sclera. I suspect the condition usually results from the incarceration of a tag of bulbar conjunctiva in the wound.

Several modifications of this method suggest themselves, namely, two or three conjunctival flaps held by the single suture or by two sutures, three separate flaps and three sutures, three flaps held by two sutures, and one large flap, not undermined, held by two or three sutures. Such modifications possibly would be worth while in special cases, for instance, where an especially large incision was indicated or if paroxysms of sneezing or coughing were expected to occur. I have not, however, yet demonstrated their value by actual trial.

PROTECTION OF THE EYES BY REFLECTING GLASSES.

By Dr. JOSEPH IMRE, Jr., University Eye Hospital, Pécs, Hungary.

(With three illustrations in the text.)

THE subject of protecting glasses has a literature, and is of interest not only to ophthalmologists but also to industrial hygienists and experts on illumination. Yet several questions concerning the influence of radiating energy on the visual organ are still awaiting a definite answer.

Thus we have no unanimous opinion as to how much light energy may reach the eye without disadvantage. On the other hand, we do not know what effect the ultra-red and the ultra-violet rays may have on the eye in addition to their disadvantageous influence, which is experimentally proven.

In other words—up to now these two important groups of radiation at the two ends of the spectrum were examined only regarding their disadvantageous influence without having their possible biological necessity taken into consideration.

We see, furthermore, that protecting glasses were studied only with regard to healthy eyes. It is very important that we should learn just what kind of rays should be kept back from the diseased eye according to the nature of the disease, and which rays can be looked upon as exerting a healing factor in certain eye diseases.

We know that the short-wave light has a good influence on tuberculous processes and that in such cases smoked glasses are not quite rational because they keep back the violet and ultra-violet rays.

This consideration awakened my interest and prompted me to investigate whether we should be satisfied with the protect-

ing glasses that have been in use up to the present time or whether we should look for something better for certain purposes.

Before reporting on my own investigations I will take the liberty of giving in brief the history of protecting glasses.

The first protecting glass was recommended 210 years ago (Chr. Hertel, 1716). These glasses were green. At the end of the 18th century statements appeared in ophthalmological literature about the rôle played by light in the etiology of eye diseases. Plenck, the first university professor of ophthalmology, wrote in 1788 that the light of the sun and of open fireplaces is the cause of many eye diseases. Beer (1817) saw for the cause of cataract formation the rays of the sun and the strong open fire beside which men worked.

The first study of the question based on statistical data was published in 1857 by Frigyes Grosz, the grandfather of Emil Grosz, the present professor of ophthalmology in the Budapest University. His data were quite in harmony with the arguments of Widmarck thirty-two years later. He was the first to point out that the ultra-violet rays are the most dangerous group of radiation for the eye.

Following Widmarck, William Schulek, the founder of the modern Hungarian ophthalmic school and professor at the Budapest University, analyzed 225 different chemical agents with regard to their absorption of ultra-violet rays.

These studies may be looked upon as classical and epoch-making in this chapter of experimental ocular physiology. Widmarck's merits are not lessened by the circumstance that the experimental studies on the etiology of cataract made us sceptical regarding the great importance of ultra-violet rays in this respect.

In order to be clear in what is to follow, I shall have to digress. The different opinions concerning the etiology of cataract may be divided into three groups. One group believes in the pathogenic importance of the short rays (Schanz, van der Hoeve, Birch-Hirschfeld). The second group explains the development of cataract through the influence of red and ultra-red rays (Crookes, Vogt and his pupils). The third group, which sees the basic cause of cataract in constitutional abnormalities and a certain predisposition, with-

out denying the possibility of other influences like radiating energy, includes Pineles, Kuwabara and Imre. Ultra-violet rays which are shorter than 300μμ do not pass the cornea and thus they cause only superficial changes. Snowblindness, the so-called Gletscherbrand, the eye inflammation of film actors, is caused by these short-wave ultra-violet rays. The pigmentation of the skin is also caused by rays of around 300μμ length.

The relatively long-wave ultra-violet rays, between 300 and 400, reach the anterior chamber but are absorbed by the lens. The absorbing ability of the lens changes with age. The lens of a 65-year-old man absorbs all the rays shorter than 418μμ, whereas the lens of a four-year-old child absorbs only those shorter than 305.5μμ. The difference may be explained by the variation of the thickness, color and consistency of the lens (Shoji).

Experiments with ultra-violet rays never resulted in cataract. Birch-Hirschfeld exposed eyes of rabbits to ultra-violet rays without producing real cataract, merely slight changes in the epithelium of the lens.

The experiments of Hess with the sparks of a Leyden jar gave positive results which may, however, be looked upon as a mild form of the so-called lightning cataract where, just as in cases of cataracta electrica, factors other than ultra-violet rays play an important rôle.

Parsons, Henderson and Martin found that energetic irradiation with ultra-violet rays *causes* a change in the function of the ciliary body with the result that hæmolysins appear in the aqueous fluid. Birch-Hirschfeld found pronounced inflammation of the ciliary body in such experiments.

The experiments and theory of Van der Hoeve make the possibility of an influence of the ultra-violet rays on the nutrition of the lens even more probable. He pointed out that the lens is optically not "empty," not homogeneous, therefore the light rays, especially the short-wave ones, suffer an "inner diffusion," or diffuse dispersion. This dispersion differs from fluorescence because through this diffusion the length of the waves does not change-contrary to fluorescence.

The ultra-violet rays around 300μμ are scattered 50 times more than the red rays around 800μμ. In other words, while

the visible rays are not much influenced by the heterogeneity of the lens, the greater part of the ultra-violet rays does not pass the lens undisturbed. Through this inner diffusion a part of the scattered ultra-violet rays reaches the epithelium of the ciliary body and in a few decades this may lead to changes in its cells and in the nutrition of the lens.

Without denying the possibility or even probability of this, I wish to emphasize the fact that other factors must have a part in the development of cataract. There are differences between cataractous and non-cataractous persons. For instance, the urine of cataractous patients contains on an average $\frac{1}{3}$ less ammonia than that of persons of the same age and under the same conditions, but without cataract, as Kuwabara and I could show sixteen years ago.

Vogt and his pupils showed that ultra-red rays and red rays between 700–1500 may cause cataract. These rays cause the cataract of glass blowers and ironworkers. They studied the question of protecting glasses and found that the best glasses, through which the heat rays cannot pass, are the iron oxydul glasses recommended by Zsigmondy more than thirty years ago. This glass, under the name of Robon glass, is produced by the Zeiss factory.

This long introduction was necessary to show in what direction the studies concerning the disadvantageous influence of visible and invisible rays lead. I do not wish to report on the experiments on animals with quartz, Röntgen and other rays, because it would lead me too far.

These early experiments prompted investigators to put the production of protecting glasses on a scientific basis.

These glasses may be arranged in three groups. In the first category we can place the common colored glasses which were and still are produced without any scientific basis and were recommended for healthy as well as diseased eyes. The second group contains those glasses which absorb the ultra-violet rays and in the third group are those which absorb the infra-red rays.

Most of the modern protecting glasses were recommended for absorption of ultra-violet rays and the well known Hygat,

Hallauer, Euphos, new Enixanthos, Crookes, and Ultrasin glasses are more or less ideal and all of them are satisfactory for this purpose. For absorption of infra-red rays Vogt recommends iron oxydul glasses. The glasses of Crookes, Number 240 with iron oxalate, or 217 with ferric carbonate, are likewise excellent. (Fig. 1.)

Glasses that are equally good for either end of the visible spectrum are not in use. Some of the Crookes glasses absorb

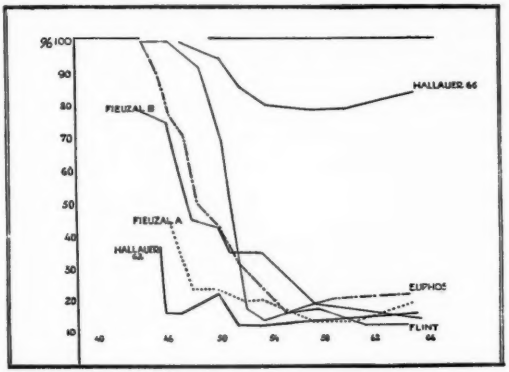


FIG. 1.

the ultra-violet rays under 390 and also absorb 35–37% of the infra-red rays, but this is not satisfactory.

The generally known protecting glasses all suffer in that they not only do not give protection against heat rays, but through the absorption even a considerable quantity of the visible rays will be lengthened and changed to heat rays and thus a decided accumulation of heat will be produced behind these glasses. This is noticeable even when not very dark protecting glasses are worn at the seashore or in the mountains.

It is very desirable that the superfluous or even harmful rays should be held back from the eyes not by absorption, but by reflection. This is also noticeable to a slight degree in every-day glasses. We can increase this reflection by glasses which have the characteristics of a mirror but at the same time let through the necessary quantity of visible rays.

It is known that very thin metallic layers are transparent, but they have also quite an extraordinary reflection-ability; the rays which do not pass through the thin metal layers not being absorbed are mostly reflected. The reflection is so strong that, for instance, a silver layer of 15 millionth millimeter looks like a regular silver mirror, although it is very transparent because it lets through 32% of the blue and violet rays between 460 and 480μμ.

A gold layer of 10μμ thickness lets through the green rays, *i.e.*, between 500–540μμ in 38% and looks in the light that falls on it like a fairly good yellow mirror.

The reflection of the ultra-violet rays may be looked upon as perfect if the layer is thicker than 15μμ. Silver is an exception because through a silver layer of 15μμ thickness there passes a considerable quantity of short wave rays. (Fig. 2.)

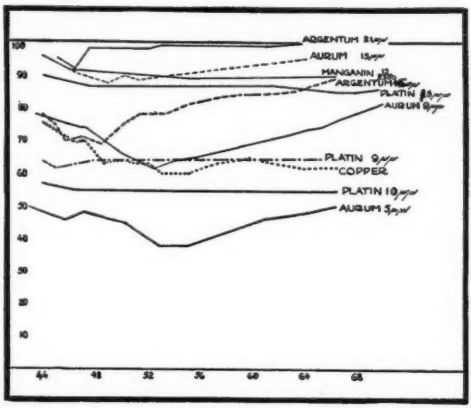


FIG. 2.

These two metals, silver and gold, have a selective transparency, gold for green, silver for blue rays. Platinum holds back the whole visible spectrum quite evenly and its color is therefore neutral gray.

As regards the heat rays which are checked by thin metal layers, we know through the studies of physicists that heat rays of about 1000μμ wave length are checked by platinum in

73%, by gold in 95% and by silver in 97%, almost exclusively by reflection. These relatively short heat rays interest us especially because of the papers of Vogt and his pupils on the question of the damaging effect of heat rays on eyes. The data concerning reflection ability of thin metal layers are to be found in the Landolt-Börnstein article.

These examinations were made with the exclusion of visible rays and thus the heat reflection was measured exclusively. I made a series of experiments, which showed to what extent the heat and visible rays, which may be lengthened by absorption to heat rays, are held back by thin metal layers.

We used as heat and light source a microscope lamp of 100 watts. We put before the lamp a glass, the walls of which were parallel and *2mm* thick, filled with water. The thickness of the water filter was *14mm*. This filter held back all the heat rays above 1500μμ wave length. These long wave heat rays do not interest us from an ophthalmological standpoint.

Eight centimeters from this filter stood the thermometer, the mercury of which was covered with soot. I used as control the increase of the temperature of the water, which was in every experiment 11° C. in ten minutes. (Fig. 3.)

The results of these experiments are shown on the third chart. This proves also that the glasses covered with thin

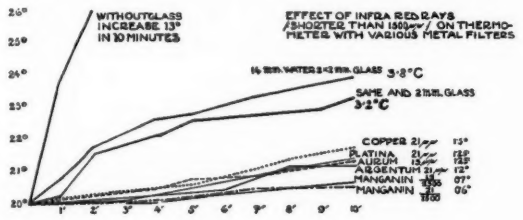


FIG. 3.

metal layers protect the eyes equally from the ultra-violet, ultra-red and in any desired degree from the visible rays. The wearing of such glasses is very agreeable, because there is no accumulation of heat behind them. This explains why these glasses cause a feeling of coolness which is quite considerable in summer sunshine.

This absence of feeling of heat around the eyes is not only subjectively agreeable for diseased eyes but also advantageous. When using the every-day smoked glasses, the accumulated heat causes a relatively quicker evaporation of tears which cover the cornea. This provokes more blinking, an increase in tear production and the inflamed eye has not the necessary rest under these glasses. They are quite naturally agreeable for the patients, because a great percentage of the irritating light rays are kept back from the eye, but they are not perfect.

This may explain why our patients prefer wearing reflecting glasses rather than any of the other well known protecting glasses. The more thorough rest secured under the reflecting glasses also explains why the most irritated eyes become quieter under them. If we return to smoked glasses, the eyes become more irritated within a day and the patients ask for a return to their metal glasses.

We gradually extended the use of these glasses in the university eye hospital and in general we order glasses with gold or platinum layers, which reflect 80 to 90% of the visible rays. For cases of very acute iritis we use platinum glasses in a thickness that reflects 98%. The 2% of visible rays is quite sufficient for the patient to go around unhindered, because the eye becomes adapted to this quantity of light within ten or fifteen minutes.

For eye diseases on a tuberculous basis or for those developing as a consequence of avitaminosis, it is best to give glasses with a silver layer, because these reflect the visible rays in any desired percentage and let through only the rays between 480–430 in greater quantity. Even the ultra-violet rays pass in considerable quantity if the silver layer is not thicker than 15.

On this and on the basis of the experiments of Lundsgaard and others we collected data concerning the effect of such filtrated radiation on certain diseases. The period of the experiments has been too short up to now to present definite conclusions, but one fact is certain: the patients feel subjectively much better because their great photophobia is checked by the filtered light and the pathologic process seems to be advantageously influenced.

All these patients use metal glasses in a light goggle frame which protects the eyes from side illumination and reflexes. The plano-parallel glasses give a disagreeably clear mirror image of the objects standing behind us, which could be useful for detectives, but may disturb others. But wearing them in the well adapted frames just mentioned, our patients walk in the garden of the clinic, go up in the solarium and are lying in their chairs for hours in the sunshine.

It is easy to imagine what a great help this is for general treatment. Even the most severe iritis cases heal at least as quickly, if not much more quickly under the necessary causal treatment than those in dark or semi-dark rooms, or by using smoked glasses. This freer treatment has quite considerable psychic advantage over and above the treatment of earlier days when we kept our patients in gloomy and dark rooms. Those patients who went through several iritis attacks notice the great difference within a few days. They claim that the reflecting glasses bring them a conspicuous relief.

It is quite another question as to how far the reflecting glasses may be used by healthy eyes. The physical qualities of these glasses make them quite excellent for use in the excessive sunshine on the seashore and when walking on snowy mountains. In such cases meniscus glasses should be used in every-day frames.

I wish to say a few words about the technical questions arising in connection with thin metal layer glasses. The one who made these glasses for me succeeded in making thin metal layers with an accuracy of one millionth millimeter. The layers are absolutely even, and when I examined them under the microscope I could not find any heterogeneity even under a magnifying power of 1500 times.

The expense of production is so low that the glasses should not cost any more than ordinary glasses.

Looking up the subject in the literature I found two recent data on thin metal layers recommended as a protection for heat rays. Both are mentioned in a paper of Schnyder (Graefe's *Archiv*, cvi., 3) where it is stated that the American Optical Co. recommended "gold plate glasses" and that the German firm of Nitsche and Günther wishes to solve the

question of heat protection of eyes with metallic eyeglasses. I could not find the original description of the American glasses and several opticians tried to supply me with such glasses, but failed.

The German firm writes to me that their glasses are brown or bluish brown, which shows that they did not work with the same metals we used. After having read this paper I visited the American Optical Co. and found that they produce gold plated glasses for welders. These in the thickness recommended for industrial purposes are not suitable for our purposes.

We examined iron, copper, manganese, etc., but found that these metals have the greatest reflection ability. The only exception is manganese, which reflects the heat rays very well, but acts on the short wave rays only if used in relatively much thicker layers than the other metals.

It seems to me very probable that a combination of several metal layers may give even more surprising results; therefore, we intend to continue our experimental examinations in the near future.

DYSTROPHIC INTRACELLULAR OPACITY OF THE CORNEAL EPITHELIUM.

By Drs. NELSON M. BLACK and F. H. HAESSLER, Milwaukee, Wisconsin.

(With eight figures on Text-Plate V.)

SUMMARY.

A HITHERTO unreported corneal lesion and its successful treatment are described. The patient had an opacity limited to the corneal epithelium, presumably due to products of faulty or slowed metabolism within the individual cells; a trophic lesion which disappeared under stimulation of moderate heat applied by means of the Shahan thermophore. The differences between this lesion and previously described non-inflammatory, avascular, superficial, corneal lesions are pointed out.

The patient, a white male, aged 25 years, was first seen in February, 1920. He stated that his right eye had been practically blind since six years old. He knew of no injury. Examination revealed myopia of 20 D. and advanced chorioretinal scars, atrophy and pigmentation. With the left eye, which was externally normal, the vision was $\frac{20}{25}$ plus.

In April, 1924, the patient returned. Three months previously the vision had suddenly become blurred and till now had been treated bi-weekly with salve and drops, but no improvement has been noted. Vision, left eye, was $\frac{20}{200}$. A superficial corneal opacity was seen, which covered roughly the area shown in Fig. 1, except that there was a clear zone between the affected portion and the limbus. Treatment with hot compresses, thiosinamine ointment, dionin and curettement of the epithelium had no effect on the lesion.

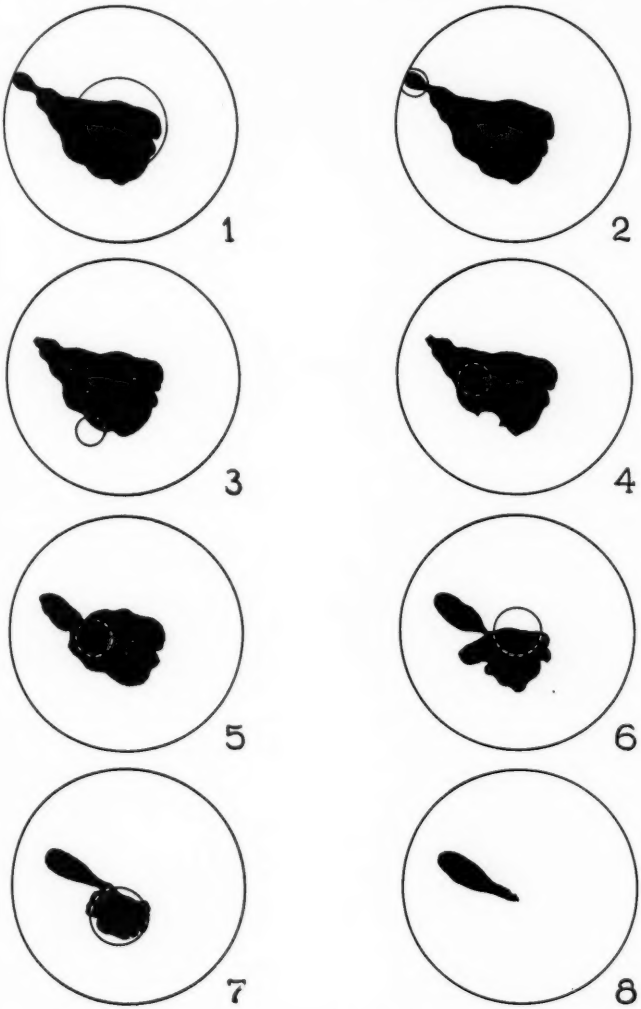
In September, 1924, a small dark area was seen and thought to be a foreign body. Examination with the slit-lamp made it clear that there was no foreign body, but merely a clear area within the limits of the opacity. An exhaustive general examination revealed no deviation from normal except some questionable teeth. These were extracted, sodium salicylate was prescribed, but the corneal lesion remained unchanged.

Application of the Shahan thermophore had been considered, but in view of the obscurity of the nature of the lesion, the uncertainty of the effect of possibly stimulating it with heat and the absence of a sound fellow eye, it took courage on the part of the patient as well as the oculist to try this form of treatment. However, as the lesion remained unchanged after a year of other therapeutic measures and the vision in the affected eye was not clear enough for efficient work, it was decided to treat cautiously a very small area outside of the pupillary region.

In the meantime Knuesel (1) had outlined his technique of examining the corneal epithelium with the slit-lamp, and a more thorough microscopic study of the cornea was undertaken preliminary to thermophore treatment. The extent of the lesion is shown in Fig. 1. Grossly, it looked precisely like an area of ground glass in an otherwise normal cornea. Examination with the slit-lamp and corneal microscope made it clear that the lesion was limited entirely to the epithelium. This tissue was neither thickened nor thinned, nor irregular in surface. With the highest power it looked granular and slightly yellowish. There was no variation in different parts of the affected area. At its edge the lesion had precisely the same appearance as at its center. There was no gradual transition from morbid to normal tissue. A sharp line divided the normal epithelium from the pathological. No changes whatsoever could be discovered in Bowman's membrane in the substantia propria or its nerves, nor in Descemet's membrane and the endothelial lining. The cornea was less sensitive to tactile stimuli within the limits of the lesion than elsewhere. Staining with fluorescin was never unequivocally successful.

It seems unlikely that a lesion which causes opacity of the corneal epithelium without changing its thickness or position, can be anywhere except in the cells themselves. A change must have taken place in the protoplasm of the epithelial cells, which makes it less transparent. Whether this is an infiltration of fat, hyaline or some other substance it is impossible to say without a microchemical

ILLUSTRATING ARTICLE OF DR. NELSON M. BLACK AND DR. F. H. HAESSLER, "DYSTROPHIC INTRACELLULAR OPACITY OF THE CORNEAL EPITHELIUM."



No. 1 is a diagram of the extent of the lesion at its height and its relation to limbus and pupil.

In each of Figs. 2 to 8 the small circle indicates the size and position of the area covered by the thermophore, and the decrease in size of the black area indicates the clearing of the opacity brought about by the application of the thermophore, as indicated in the preceding figure.

The diagrams are accurate representations of the size and shape of the lesion on the following dates:

No. 1—December	1, 1925.	No. 5—December	14, 1925.
No. 2— "	1, 1925.	No. 6— "	18, 1925.
No. 3— "	7, 1925.	No. 7—January	4, 1926.
No. 4— "	11, 1925.	No. 8— "	13, 1926.

study of some excised tissue, which did not seem justifiable in this case.

Application of the Shahan thermophore did not destroy the epithelium. As would be expected from mere mechanical pressure, a crater was formed, in the center of which the tissue was thinned and at its edge there was a heaping up of tissue. The heating, however, did not produce immediate optical transparence of the tissue, nor did it produce a white opacity as does a necrotizing agent. Twenty-four hours later the surface was again smooth and some small part of the involved epithelium was again clear. However, as will be seen from the text figure, it was not always precisely the treated area that was cleared by the application of the thermophore. In other words, it was not a mechanical "ironing away" of the epithelial defect. In the diagram the black area is an accurate representation of the extent of the opacity and the circle indicates the position of application of the thermophore at that date.

The effect of each treatment can be seen from the disappearance of black area in the succeeding figure. A final application of a large thermophore surface removed the remaining club-shaped area of opacity. With a correcting glass the vision is $\frac{6}{6}$ and he reads D equals 0.37.

Pathogenesis.

In the course of a year much of the corneal epithelium is presumably cast off and replaced by new cells growing up from below. However, in one particular area of this cornea an opacity was constantly present. Hence some process must have been active, which produced the same change in the newly growing cells as in the older ones. The fact that one application of very moderate thermic stimulus should produce disappearance of the opacity, usually in the area stimulated, but sometimes in adjacent areas, suggests that the lesion is a trophic one. The heat presumably stimulated the sluggish metabolism so that the droplets or granules abnormally present in the protoplasm were absorbed, destroyed or changed in composition.

Differential Diagnosis.

We have then to differentiate a very superficial, avascular, corneal opacity in an eye entirely free from inflammatory

manifestation, non-familial and remaining unchanged for months, from other lesions having some of these characteristics. We will briefly review the characteristics of these entities, so that it may become clear that the dystrophic, intracellular, epithelial opacity does not merely differ in accidental details, but is the manifestation of an essentially different morbid process.

Keratitis epithelialis punctata, of which five cases were described by Koeppe (2), consists of discrete and confluent, minute and greyish, sharply defined and punctate opacities limited to the epithelium, but visible only with high magnification in slit-lamp illumination. Sometimes the superficial stroma layers are slightly edematous. Grossly the eyes seemed normal except for slight hyperæmia and after a few days or weeks subjective irritation, as well as punctate opacities, disappear without treatment. Kraupa has observed similar cases.

Keratitis punctata superficialis would hardly be confused. The signs of severe irritation, alone, distinguish this disease from the case observed by us, and examination with the binocular microscope leaves no doubt that the multiple, discrete, punctate opacities are subepithelial.

Herpes corneæ, too, of which keratitis epithelialis superficialis is considered a variant by some observers, manifests itself by severe irritation and its minute blebs could hardly be confused with the dull, lusterless opacity of the dystrophic, intracellular opacity.

Nodular opacity of the cornea, of Groenouw, occurs in eyes free from all irritative manifestations. It always occurs bilaterally and the corneal surface is very uneven, with many small protuberant nodules. The stroma is not free from punctate opacities. Histologically there is swelling and destruction of corneal lamellæ, the deposition of amorphous acidophilic masses under the epithelium and a basophilic substance in the stroma. In one case the tissue beneath the epithelium was normal and the process involved only one eye.

Reticular opacity of the cornea (Haab, Dimmer, Fehr, Meller, Stähle and others) consists essentially of linear opacities over which the epithelium is raised in the central, corneal area and accompanied by transient redness and irrita-

tion. The epithelium itself becomes opaque. The manifestations are the result of hyaline change which takes place in the deepest epithelial strata and also involves Bowman's membrane.

In the epithelial dystrophy of Fuchs the lesion begins centrally, there are irregular eminences on the corneal surface and a new, formed substance is deposited under the epithelium.

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REPORT OF A CASE OF FLAT (INFILTRATING) MELANOSARCOMA OF THE CHOROID.[1]

By Dr. JAMES W. SMITH, New York.

(With two illustrations on Text-Plate VI.)

THE following case and photomicrograph is presented because of the rare incidence of this type of choroidal sarcoma. Fuchs in 1882 reported three cases and is usually given credit for distinguishing between diffuse and circumscribed sarcomata of the uveal tract, although De Wecker mentioned two cases in the *Graefe-Saemisch Handbuch* of 1876. Mitvalsky reported two cases in 1894 and first used the very descriptive term "Flächensarcom." Very few cases have appeared in American ophthalmologic literature (Alling, A. Knapp, Woolley, Goldberg, Wescott and Lilley), and the total number reported in 1921 was forty-five. The most important contributions to and reviews of this subject in the last decade have been made by Parsons, A. Knapp and Fuchs.

Previous History.—Mrs. Paulina L., age 26, was first seen by me in the clinic of Dr. Martin Cohen at the Post Graduate Hospital on August 4, 1925. The patient stated that during the past seven years there had been a progressive loss of vision in the right eye resulting in complete blindness two and one-half years ago, in the seventh month of pregnancy. Prior to this time she had undergone treatment for ocular tuberculosis. Since the onset of blindness the right eye had been injected and painful and the patient observed that the globe was protruding. For nine months preceding her admission there had been intermittent attacks of severe orbital pain.

[1] Read before the Section on Ophthalmology of the New York Academy of Medicine, January 18, 1926.

ILLUSTRATING ARTICLE OF DR. JAMES W. SMITH, "REPORT OF A CASE OF FLAT (INFILTRATING) MELANOSARCOMA OF THE CHOROID."

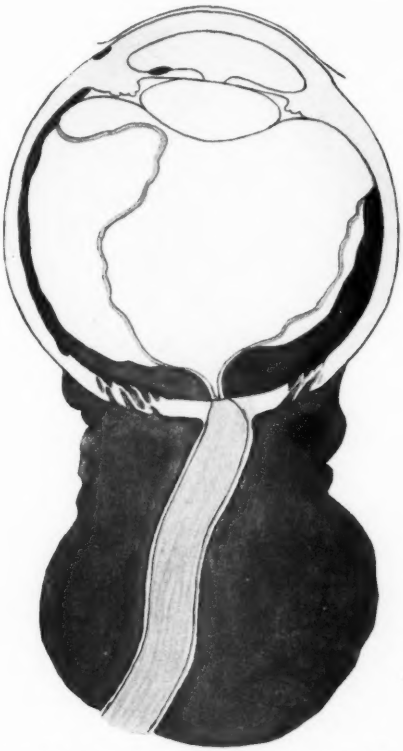

FIG. 1. Schematic sketch of the topography of the tumor.

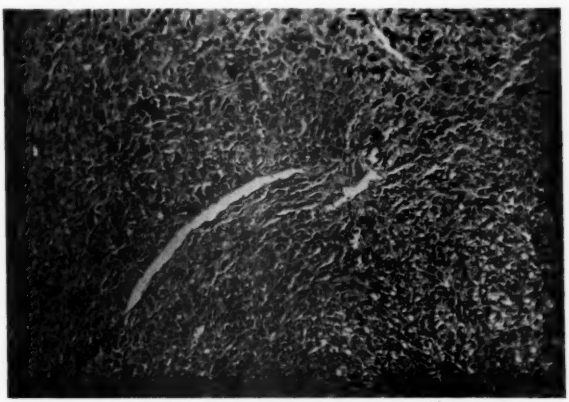

FIG. 2. Section through tumor showing its typical structure. In this area the melanin-containing cells are sparsely distributed.

UNIV. OF MICH.

Clinical Examination.—V. O. D. Complete blindness. V. O. S. $\frac{10}{20}$. The cornea and anterior chamber of the right eye were negative. The limbus showed an injection of the ciliary vessels. Exophthalmus of moderate degree, not measured with the exophthalmometer. Tension 55*mm*, left eye 17, with Schiotz tonometer. The pupil was fixed in dilatation and a large area of gray detachment seen inferonasally. The fundus could not be examined. The nasal half of the globe could not be transilluminated. Ocular movements limited in the fields of the internal rectus, superior oblique and inferior oblique muscles. Examination of the left eye negative. The diagnosis of sarcoma of the choroid with extension to the orbit was made based on the paresis of the three ocular muscles. Immediate exenteration of the orbit was advised.

Operation.—Two days after admission exenteration of the right orbit was performed. The periosteum on the temporal, superior, inferior and anterior third of the nasal surface was easily freed. Considerable difficulty was encountered along the nasal aspect of the orbit posteriorly and the patient reacted unfavorably to dissection near the apex. The nasal surface of the globe appeared extended posteriorly into a pear-shaped tumor mass which completely ensheathed the optic nerve. The orbit was tightly packed and the patient returned to the ward in surgical shock.

The patient remained in shock for three days with unusual reduction in the sphygmomanometric readings; August 7th, systolic 80, diastolic 12; August 8th, $\frac{88}{50}$, and on August 10th $\frac{110}{70}$. The further course postoperatively was uneventful and the patient was discharged on the tenth day and referred to the Memorial Hospital for radium treatment.

Preliminary Pathological Report (Dr. Paul Klemperer).—Gross: specimen is bulb with conjunctiva, fat tissue and muscle tissue adherent. The bulb is collapsed. Attached to the fundus there is a yellowish-gray mass which has been separated by a cut from another mass which is closely attached to the optic nerve. The measurements of the collapsed bulb are approximately 30 x 20 x 20*mm*. The yellowish-gray tumor mass at the central part of the nerve measures, including the nerve, 25 x 20 x 15*mm*. The tumor can be separated without difficulty from the surrounding fat tissue. Gross sections made through the optic nerve and the tumor show that the optic nerve is ensheathed on both sides by the tumor (Fig. 1). Section through the eye shows that the tumor perforates the sclera and invades the surrounding tissue.

Microscopic.—Sections were taken from the tumor in-

volving the nerve, without nerve however. The tumor is composed of large polygonal cells with rather large pale nuclei with distinct nucleoli which show an alveolar arrangement. Mitotic figures are rather frequent and there are areas of necrosis. One portion shows brown pigment in the tumor cells as well as in the cells of the stroma. The pigment does not give iron reaction and is apparently melanin (Fig. 2).

Sections from the surrounding muscle tissue do not show invasion by tumor cells.

Diagnosis.—Melano-blastoma.

Final Pathological Report (Dr. Louise H. Meeker).—The choroid is entirely replaced by a tumor which causes it to be diffusely and irregularly thickened to several times its normal thickness, especially at the fundus. For the most part the chorio-capillaris can be still recognized. The sclera and the choroid are firmly bound together by the invading tumor cells which dissect the scleral lamellæ apart and extend along the vessel sheaths to the exterior of the eye. Extension has taken place along all the posterior ciliary vessels and a tumor about the size of the eyeball surrounds the optic nerve. The extension along the optic nerve has obliterated all traces of the dura and arachnoid. The venæ vorticosæ also are surrounded and invaded by the tumor.

The anterior segment of the eye shows extensive involvement. From the choroid the tumor grows forward into the ciliary body for some distance and is seen extending along the blood vessels of the anterior ciliary system out through the sclera. At least one vessel at the limbus is crowded with pigment-bearing cells and pigment-bearing cells lie on Descemet's membrane and also fill the spaces of the filtration angle and Schlemm's canal.

The iris has a thin coating of tumor, highly vascular, on its anterior surface, unpigmented however.

A mild inflammatory reaction is evident at the limbus and ciliary body where the blood vessels generally contain many polynuclear leucocytes and polynuclear leucocytes are scattered sparsely through the corneal stroma. The chambers of the eye are empty and the vitreous space is obliterated except for a small portion. The lens is not remarkable except for adhesions of the iris pigment epithelium.

The tumor replacing the choroid is composed of rounded cells for the most part but in some areas they are plainly spindle cells of the usual choroid sarcoma type, that is, interlacing compact bundles of spindle cells. The amount of pigment varies greatly in different portions of the tumor. As a rule there is little pigment but within the bulb espe-

cially in areas showing beginning degeneration there is much pigment. The extra-bulbar extensions of the tumor are mostly without pigment. The coating of tumor on the iris is unpigmented. Mitotic figures are fairly frequent and there are areas of degeneration and beginning necrosis. The diffuse distribution and the manner of growth of this tumor is typical of the flat sarcomas of the choroid with which this tumor is to be classed. We have especially the early penetration through the emissaria of the sclera and a large tumor behind the bulb producing early exophthalmus. Moreover we have no circumscribed tumor mass as in the usual sarcomas of the choroid.

Diagnosis.—Melano-blastoma, more commonly designated "Sarcoma Choroidæ Diffusum."

Memorial Hospital Report.—Dr. Douglas Quick reports that on "August 21st patient was treated by the use of 2500 millicurie hours radium emanation, filtered by 2 millimeters of glass and placed at a distance of 3 centimeters directly in front of the orbit and a similar dose through the region of the temple, that is 'getting at it from the side.' On December 2, 1925, the dose from the front was repeated and on December 4th that from the side was repeated."

Present Status.—The orbital floor has been filled in since the exenteration by a substance almost bony in its resistance to palpation which impressed me as being proliferating tumor tissue. Dr. Quick however views this tissue as evidence of an extreme degree of fibrosis with a certain amount of calcification and that its formation was stimulated in part at least by the radium that was used last August.

The Roentgen ray report of the Post Graduate Hospital (December 21, 1925), states that "the illumination is somewhat diminished in the right ethmoidal region as if from membranous thickening, otherwise no gross abnormality is noted. There is no evidence of a destructive process." The report of the Memorial Hospital (January 15, 1926) states that, "Stereoscopic films of the orbits fail to reveal any evidence of alteration in bone density. No evidence of bone destruction is seen. The right orbit, however, when compared with the left seems somewhat more cloudy and the appearance suggests a uniform increase in density of the soft parts, possibly fibrotic in nature."

X-ray Examination of the Optic Canals and Sella Turcica.—January 29, 1926. "The left optic canal is normal in size, shape and structure of its walls.

"The right optic canal shows thinning and irregularity of its walls and an enlargement of 20 and 33% of the two principal diameters.

"The calculated diameters of the optic canals are:

Left	3.6 x 4.5*mm*
Right	4.3 x 6.0*mm*
Enlargement	20% & 33.3%

The thickness of the bridge which separates the optic canal from the sphenoidal fissure is 3.0*mm* on the left side and 1.8*mm* on the right. This, together with the irregularity of the walls of the right optic canal, indicates that we are not dealing with a normal variation in size, but with an actual enlargement.

"The anterior clinoid processes are thick and symmetrical. The sella turcica is normal.

"There is no evidence of extension of the pathological process beyond the optic canal (intracranially), so far as the X-ray is concerned. A later examination is, of course, desirable.

Harry A. Goalwin, M.D."

A thorough physical examination made on January 15th reveals only the presence of a slight cardiac irregularity in the form of occasional extra systoles. The entire examination was otherwise negative. There was no suggestive evidence of tumor metastasis.

Comment.

This case is interesting pathologically because of its difference in appearance and growth from the usual circumscribed sarcomas of the choroid. The duration of the disease in this patient follows the classical description which varies from seven months to ten years. The age incidence is not constant, the youngest case reported being twelve years of age and the oldest seventy-two.

Further studies will be made on this patient in an effort to explain the presence of a highly malignant tumor for at least two and one half years (and possibly seven years), with orbital involvement, with no definite evidence six months after exenteration of local extension or generalized metastasis.

An interesting reference to the difficulty sometimes encountered in the clinical diagnosis of flat sarcomas of the choroid is made by Parsons in his record of thirty-five cases of diffuse sarcoma of the uveal tract (*Archives of Ophthalmology*, Vol. 33,

1904), when he reported that "a number of the eyes were enucleated because of absolute glaucoma and the tumor discovered in the laboratory."

I desire to thank the pathological department of the Post Graduate Hospital and especially Dr. Louise H. Meeker for the report and photomicrographs of this case.

THE RELATION OF THE NASAL GANGLION TO GLAUCOMA.

By Dr. HIRAM BYRD, Bradenton, Fla.

IN 1923 the writer assembled evidence that the nasal ganglion is at the bottom of glaucoma. That evidence can best be summarized by quoting the introductory paragraphs from Felix Lagrange, "'Du Glaucoma et de L'Hypotonie," which may be considered the last word on this subject. Lagrange takes the position that in glaucoma "there is a neuropathy producing a hypersecretion, as first postulated by Donders, leading to increased tension when, as shown by Knies, the anterior chamber angle becomes blocked."

So far all have agreed that glaucoma is a hypertension. This hypertension is initiated by a neuropathy. But it results in hypertension only when the anterior chamber angle becomes blocked. The two necessary conditions of glaucoma then are increased secretion and poor drainage.

The increased secretion is due to neuropathy, but no one has as yet designated the particular nerve that has gone awry. If we could place a finger on that nerve it would be a distinct advancement. That is the object of this paper.

Morax and Girard, studying the intraocular tension in animals, observed that if the needle of the canula came in contact with the iris the tension was so disturbed that the animal had to be abandoned and a fresh one taken. Such was the effect on tension of a stimulus applied to the iris.

Migitot later measured the disturbance of tension caused by local stimulation of the iris. Using cats for this purpose, he found that the normal tension is 30*mm* of mercury, but in three minutes after the needle of the canula touched the

iris the tension mounted up to *42mm*, and in seven minutes reached a maximum of *56mm* of mercury.

Turning now from the eye to the nose, Sluder, of St. Louis, reports that in a particular case after cocainizing the nasal ganglion on the right side, the eye lid drooped and the pupil contracted to about half the size of its fellow. This causes him to remark: "The case presents this interesting phenomenon; that through the nasal ganglion in this case must pass a large part of the sympathetic supply which goes for the elevation of the eyelid and to the pupil."

To me the interesting point is the proof that a *nerve impulse* from the nasal ganglion has a direct path to the pupil. Remembering the anatomy, it will be seen that such an impulse must traverse the structure that secretes the aqueous, namely, the ciliary body. Here then we have Meckel's ganglion connected with two secreting structures about the eye—the one external, the lacrymal gland; and the other internal, the ciliary body. Stimulation of the ganglion excites excessive secretion of the lacrymal gland, and anatomically and physiologically we have every reason to expect a similar effect upon the ciliary body.

Since that time three important observations have been made, namely: 1. It has been shown that "pathological currents" may originate in one locus, as a focus of infection, or other pathology, and be transmitted to—,and register in another locus, other than the brain, more or less remote from the locus of origin, and that a physiological disturbance is set up at the locus of registration.

2. It has also been shown that pathological currents find their way to and register in all the following structures about the eye: A. lachrymal gland; B. conjunctiva; C. cornea; D. ciliary body and iris; E. peri-ocular structures.

3. It has also been shown that these pathological currents, reach the eye *only via the nasal ganglion of the same side*, and that they can uniformly be intercepted at the ganglion by cocainizing it.

There is occasionally encountered a deep pain "behind the eye" associated with pain in the temples, that seems to come via some other route than the nasal ganglion, and cannot be intercepted at the ganglion.

In such cases, however, the eye ball is not affected, the tension does not increase, nor is there any congestion of the conjunctiva, and no unusual lacrymation, and no photophobia, so that there is little or no grounds for associating this with the eye.

Summing up all that has gone before we have the following syllogism, to wit:

1. Irritation of the iris results in hypertension.
2. Pathological currents set up an irritation at the locus of registration.
3. Pathological currents may and do register in the iris.
4. Such pathological currents reach the iris via the nasal ganglion of the same side, where they can be intercepted by cocainizing the ganglion.
5. Interception of the pathological currents relieves the iris of its irritation, and thus withdraws the *cause* of the hypertension.
6. Hence the conclusion is irresistible that cocainizing the nasal ganglion of the same side in the case of glaucoma should give temporary relief from the hypertension, and that injecting it with alcohol, after Sluder, should give permanent relief.

It now remains to see how this works out in actual practice. Again quoting from the article above referred to:

> Mrs. A., 33 years of age, has eyes with poor anatomical drainage. The irides are cupped forward like saucer bottoms. Since this is a defect of anatomy, it is assumed that it has existed from birth. Yet she gives no history of eye trouble till three years ago. During the first thirty years of life she was a potential case of glaucoma, only waiting for what Felix Lagrange calls a "neuro-pathology" but which in this connection is called a "pathological current," to stimulate hyper-secretion. Three years ago the thing happened. Both eyes became involved in a glaucomatous attack, from which she has suffered ever since. The tension is always high, but varies from day to day, some days being almost normal. It is always aggravated by a "cold in the head."

Both pain and tension can be relieved by either of two procedures: either by cocainization of the nasal ganglion, or by the instillation of one half per cent. eserine in the eye.

Quoting further:

> Mrs. M., 28 years of age, has eyes with normally good drainage, but she has a pathological nose. . . . Recently she reported for help for her poor vision. Since the tension of the eye seemed normal, atropine was used for refraction. Forty-eight hours later an acute high tension developed in both eyes, accompanied by severe pain. "Cocainization of the nasal ganglia promptly relieved both the pain and the high tension.

> CASE F., of Palm Beach reported with severe pain in left eye, pupil dilated to three quarters maximum, and adherent to lens, tension high and vision blurred. Diagnosis, acute glaucoma of ten days standing.
>
> The nasal ganglion was cocainized with the result that the eye was made comfortable, only still sore to touch, and the tension still unabated.

> During the next four weeks the nasal ganglion was cocainized with a frequency corresponding to the severity of the pain. At its worst, cocainization kept the eye comfortable for about four hours. Since eserine failed to contract the pupil in the slightest, it was abandoned, and the only treatment consisted of keeping the pathological current cut off at the nasal ganglion. Gradually the eye improved, and finally was dismissed with $\frac{20}{30}$ vision, and no discomfort. The pupil was still three quarters dilated, but with a tinted glass to subdue the light, this did not seem to distress him. Last seen a year afterward, the eye was comfortable and the vision $\frac{20}{20}$.

At the time this case was under observation I was not familiar with the technic of nasal ganglion injection, and accordingly did not undertake it. But had that been done, I feel sure the course of the disease would have been much shortened. The pupil being adherent to the lens of course left one helpless in the matter of improving drainage. The only therapeutic measure therefore resorted to was the interception of the current at the ganglion.

> CASE O., of Bradenton, Fla., reported with both eyes acutely inflamed, painful almost beyond endurance, tension

high, and pupils about three quarters dilated. Diagnosis acute glaucoma, three days standing. The nasal ganglia were at once cocainized and pilocarpine instilled into eyes. The distress abated, but was not entirely relieved. From this time forward the ganglia were cocainized every four hours for 24 hours and in that time the distress was quite gone and the pupils fairly well contracted.

He returned to work (he has a clerical position) in about four days, but as soon as he began to use his eyes, they began to distress him again and it was about a week before he could back regularly into harness. In the meantime the ganglia had to be cocainized a number of times.

For four or five weeks afterwards it was necessary to cocainize the ganglia occasionally, and to keep up the use of pilocarpine. Indeed he keeps his pilocarpine to this good day, now about a year and a half after the incident. But it has been several months since the ganglia were cocainized.

This patient was a sufferer from "rheumatism," involving his back, hips, and thighs. Cocainization of the ganglia, it was found, would relieve the rheumatic pains. Also when the rheumatism was at its worst, then the eyes had a strong tendency to flare up—the pupils enlarging, the tension increasing and pain supervening.

If at the time of this attack the nasal ganglia had been injected with alcohol—a sort of permanent cocainization if you choose—recovery, it is safe to assert would have been much more prompt. We have it in mind to inject nasal ganglia if the condition of the eyes ever calls for it.

CASE P., aged sixty. Taken with great pain (right eye) on Saturday night. Attending physician administered morphine hyperdermically, but with only partial relief. The following morning the pupil was found to be under about three quarters dilation, the eye very hard, extremely sensitive to touch, and the entire region round about in great pain.

Cocainization of the right nasal ganglion made the patient relatively comfortable in say five minutes. Pilocarpine was instilled into eye. Repeated cocainizations and the continued use of pilocarpine found the patient better and on Tuesday, she was all but normal.

A few other cases would be but repetitions of these, and accordingly are not reported. But with the evidence at hand,

the writer confidently believes that when these facts are appreciated, and the treatment of glaucoma directed to intercepting the pathological current at the nasal ganglion and at the same time promoting drainage, that such operative measures as iridectomy and trephining will be a thing of the past. In no instance yet has this method of attack been a failure, even when the pupil was dilated and bound down to the lens, so that the only therapeutic measure consisted of intercepting the pathological current at the nasal ganglion.

Since writing the above, the following case turned up and was presented at the Manatee County Medical Society, June 8, 1926.

CASE P. W., colored, age about 45. First seen six months ago, at which time the following facts were noted: "Patient was in a dynamite explosion about a year ago, at which time the right eye was destroyed, and the left damaged so that he was under treatment for several weeks. At present time the left eye has advanced cataract, probably of traumatic origin."

Next seen, May 31st, 1926, at which time the following notes were made: Left eye inflamed, weeping, extremely sensitive to pressure, whole side of head aching; photophobia, although status of vision is light perception but not projection. Pupil dilated to about three quarters, and intraocular tension very high. D agnosis: Acute glaucoma of about a week's duration. Treatment: The nasal ganglion cocainized whereupon the distress in the side of the head, and in the eye proper abated, within five minutes. No other symptom affected. Pilocarpine instilled into eye and patient requested to report next day.

June 1st, patient has been comfortable from the time he left office the previous day till in the night when his eye began to ache again and the side of his head as before. Took aspirin but did not get relief.

At this time it could not be determined if the intraocular tension had abated in the slightest but it is not believed that it had. The inflammation, the weeping, the photophobia, and the distress in the eye proper and the side of the head seemed as great as the day before. The pupil had contracted ever so little, but it was manifest that the iris was firmly adherent to the lens.

On this occasion the nasal ganglion was cocainized with one grain of cocaine, which relieved the pain of the eye and the distress of the side of the head, and brought the patient to a state of relative comfort. The nasal ganglion was now injected with seven minims of 95% alcohol, with 5% carbolic acid. Patient complained of the operation being very painful, but the pain seemed limited to the region of the needle puncture in the nose. After some ten minutes the pain abated and the patient was comfortable again. The pilocarpine was continued and the patient requested to report the next day, when he stated that he had been comfortable ever since leaving the office, and that he thought he could see light a little better. The inflammation about the eye was markedly abated, the intraocular tension seemed easing down a little. The pupil was unchanged. During the following week, there was an unbroken period of improvement and when presented to the Medical Society on the evening of the 6th the only traces of the previous glaucoma were the adherent iris, and possibly still a trace of hypertension, although there was not uniformity of opinion on this point, and in the absence of a tonometer record it is impossible to make any positive assertion. Certain it is that if there was still any hypertension, it was very slight indeed. Thus was the glaucomatous process completely arrested, and the only discernible evidence of its having existed, was the adherent iris. On October 4th there had been no return of symptoms.

REPORT OF THE SECTION ON OPHTHALMOLOGY, NEW YORK ACADEMY OF MEDICINE.

By Dr. THOMAS HAYES CURTIN, Secretary.

MEETING OF NOVEMBER 20, 1926. DR. CLYDE E. MCDANNALD, CHAIRMAN.

Dr. Elias J. Marsh presented a case of **Posterior lenticonus with report of slit-lamp findings.** (To be published in full.)

Discussion: Dr. Knapp asked if Dr. Marsh was able to demonstrate a posterior displacement of the nucleus of the lens.

Dr. Marsh answered that the nucleus was apparently in its normal position. In false lenticonus the nucleus is pushed backwards and he saw such a case in Dr. Schoenberg's clinic at the Knapp Memorial Hospital some years ago.

Dr. Mark J. Schoenberg presented a case of **Detachment of the anterior layers of the iris of traumatic origin.** Patient 51 years of age, a tall, broadly built, muscular man, weighing 270 pounds, performing high diving feats at fairs and circuses, came to the Knapp Memorial in January, 1926, complaining of failing vision in his left and only seeing eye. The trouble began four months previously after a dive from a height of about 104 feet into a small shallow tank. When he came out of the tank he could not see. Within a short time his vision returned in the lower part of the field, but he could not see objects situated above the level of his head. Within a week his sight improved slightly. His right eye had been injured twenty-three years ago during a high dive and remained totally blind from a luxated lens and detachment of the retina.

The examination of the right eye revealed an atropic globe following irido-cyclitis. The left eye had a moderately dilated

pupil, deeply excavated optic disk, large patch of atrophy of the choroid and retina, surrounded by pigment, in the lower portion of the fundus, and a tension of 40*mm* (Schiotz).

The anterior layers on the temporal side were separated in places from the posterior layers. This separation appeared as if the various strands constituting the parenchyma, running mostly in a radial direction, were torn off so that they were floating freely in the aqueous humor. Some of these strands appeared like irregular shreds of tissue, attached to the iris by thin pedicles, covered by an extremely fine veil-like tissue. Other strands were less deformed and their free extremities appeared covered by pigment. In some portions the iris showed cracks or dehiscences, fusiform in shape, going through its entire thickness. By transillumination these openings appeared like illuminated little windows. The general impression gained with a magnifying loupe was as if the aqueous was filled with miniature grayish-blue seaweed. The pigment border of the pupil showed no tear. No causal relation could be made between the existing glaucoma and this lesion in the iris. It is very improbable that the iris shreds should have caused an obstruction of the angle and increased intraocular pressure. The slit-lamp examination showed the angle quite free from these strands. Furthermore, if the iris tissue and crypts have something to do with the drainage of the aqueous, this should be rather facilitated by the teased and rarefied iris tissue. The vision remained $\frac{20}{100}$ during 11 months, while patient remained under observation. Pilocarpine reduced the tension to 25*mm* Hg (Schiotz) and the pupil was contracted.

There was nothing relevant in the personal or family history that could throw any light upon the patient's eye condition. The most plausible explanation of this separation of the anterior layers of the iris is that the sudden trauma caused by awkward high diving must have suddenly forced the aqueous into the iris tissue, tearing apart the strands of its parenchyma and going through the entire thickness of the iris in some places. It is even possible that this was the starting point for his increased tension.

DISCUSSION: DR. FRIEDENBERG said that two facts must be considered in the case: 1, traumatism and 2, glaucoma.

He believed that the glaucomatous condition is the more important factor. In addition to shredding off of the iris in a few places, there is a complete loss of iris fibers in other spots.

DR. IMRE: In the two cases which I have observed there were peculiar floating shreds on the iris, some straight and some cork-screw shape; one in a patient 94 years of age, and in the other, an old woman of 92, there was a sharp segment of the iris involved; the rest of the iris was normal. Movements of the eyes gave undulations of the detached part of the iris.

The paper of the evening entitled **On protecting glasses with demonstrations** was read by Professor JOSEPH IMRE, Pécs, Hungary (by invitation). (Published in full in this issue.)

DISCUSSION: DR. KNAPP asked whether the vision was disturbed by these metallic glasses.

DR. IMRE: They do not disturb vision, but they would not be good for constant work with normal eyes.

DR. TYSON enquired on the effect in spring catarrh.

DR. IMRE had formed no definite opinion on this.

DISCUSSION ON MIGRAINE.

ANNUAL MEETING 1926. BRITISH MEDICAL ASSOCIATION.

REPORTED BY MR. P. G. DOYNE, LONDON.

DR. EDWIN BRAMWELL, in opening the discussion, pointed out that paroxysmal periodic headaches occur in certain organic conditions, such as brain tumor, cirrhosis of the kidneys, etc., exceptionally, as a late syphilitic manifestation.

There are four types of classical migraine attack—

(1) Simple hemicrania.

(2) Sick headache with vomiting and nausea.

(3) Blind headache with marked preceding and accompanying visual phenomena.

(4) A type with wider implication of the sensorium, with alteration of the sensibility of the extremities, disordered speech, confusion of ideas and impairment of memory.

Epilepsy and migraine have certain points in common, but the resemblance is superficial, and the two disorders are rarely met with in combination.

Migraine is a common condition. Statistics show that it usually manifests itself before the age of twenty and is slightly more common in females. A neuropathic heredity can be found in the majority of cases. Transmission through the mother is more common than through the father. An attack of migraine can often be attributed to an exciting cause, such as mental fatigue, exhaustion, eye strain, menstruation, alcohol, exposure, etc. Prolonged anxiety and worry are also definite exciting causes. With regard to eye strain Bramwell is satisfied that it is an important exciting factor, but finds that the correction of a refractive error rarely produces a cure.

In considering the manner in which the symptoms of the migraine attack are determined, Bramwell submits "that the symptoms of the migraine attack are to be accounted for by localized spasm and subsequent relaxation of the meningeal or cerebral arteries, or of both in varying degrees, the prodromal symptoms resulting from the spasm of the cerebral arteries, while the headache is attributable to the reëstablishment of the circulation in and the engorgement of the meningeal arteries."

With regard to the mode of action of the exciting factors of an attack, it would seem possible that endocrine disturbance, induced possibly by physical or mental exhaustion, may play a part in producing vascular disturbance in the cerebral vessels by means of the sympathetic, despite the fact that there is no proof of a vasomotor mechanism for the cerebral vessels. Further etiological considerations are suggested by the fact that often several members of a family show the same type of migrainous attacks and it is suggested that anatomical factors, such as relative stenosis of the foramen of Monro, relative disparity between the size of the brain and the cranial capacity, a small or enclosed sella, may be transmitted hereditary factors.

With regard to treatment, careful examination of the patient, both physically and mentally, with special inquiry into conditions which tend to precipitate an attack, so as to plan to avoid them, and, most important, general measures to improve the health and to avoid bodily fatigue and brain fag, should be advised. With regard to drugs, luminal in dose of 1½ grains at bed-time gives the best results.

Mr. McMULLEN, speaking from the standpoint of the ophthalmologist, said that visual disturbances were the commonest subjective sensory phenomenon in migraine. He considered that if the history were carefully inquired into, it would be found that visual phenomena occurred in 50% at least of all cases of migraine. He pointed out how difficult it was for a migrainous patient to describe these visual phenomena in detail. He thought that a constant feature in the course of, and at some moment in the visual phenomena of migraine was a loss of sight in some part of the field to a greater or less extent. In very few cases is the scotoma

definitely monocular, and very rarely is it central, involving the fixation point.

Referring to ophthalmoplegic migraine, he said that the associated headache was usually very severe and lasted longer than usual. The paralysis affects the 3rd nerve in the majority of cases, though the 4th and 6th might be involved. The paralysis passes off gradually, and attacks occur at long intervals. Patients may suffer from ordinary migraine for years before developing ophthalmoplegic migraine. The visual cortex is the most probable site of the disturbance causing the visual symptoms of migraine, though when monocular scotoma occurs it must be assumed that the optic nerve was the site of the disturbance. Examination of the fundi during an attack usually reveals nothing. With regard to ophthalmoplegic migraine, though in many cases there may be some definite focal lesion involving the root of the 3rd nerve, there are many cases which, from their clinical features, must be regarded as a specially severe form of migraine.

McMullen thinks that the immediate cause of the migrainous paroxysm is probably vascular, and refers to a case in which migraine alternated with an urticaria. He thinks that there is often an hereditary factor, and feels that the most workable hypothesis for migraine is the possession of an "inherent permanent underlying state of instability, often inherited, of some nervous mechanism, possibly the vasomotor system," and, given this inherent state, the actual development of the migraine attack may be due to worry, eye-strain, toxæmia, psychological repressions, etc. He thinks that writers are too inclined to stress one or other of the exciting factors according to their experience, instead of taking the broader view.

Dr. Walter Timme thought that migraine was a name which possibly covers many conditions. The type with intratemporal pain interested him most. He suspected the pituitary body as the seat of the disturbance. These attacks usually began at a time when genital development had reached its maturity and were induced by physical and mental fatigue, sexual excess, etc. X-rays of the sella turcica of these patients usually showed erosion of the cavity. The enlargement of the pituitary gland which produced these erosions was prob-

ably due to a demand upon it for hyperactivity to compensate for various fatigue states. He believed that such subjects had originally a congenitally small sella turcica and that a hyperplasia of the pituitary occurred, causing the bony erosions, and that later on in life the subsidence of the symptoms was in part due to erosions becoming sufficient to allow the sella to house the enlarged gland.

Dr. Gordon Holmes felt sure that migraine was a clinical entity with a constant and uniform pathological basis. He did not see how a hyperplasia of the pituitary could produce symptoms which developed rapidly and passed off in 4 or 5 hours. He did not find much evidence in favor of the vasomotor theory of the disease. Vascular spasm must be due to some substance circulating in the blood, but the migrainous symptoms must be due to some local change in the brain and innervation of the cerebral blood vessels had up to now never been proved. It seemed to him that the symptoms of migraine could be most easily explained by the assumption of a local swelling of the brain due to a local and temporary alteration in the osmotic relations between the brain substance and fluid with which it was bathed. He had found that the correction of refractive errors had small influence only upon the condition.

Dr. Gordon said that there seemed to be some inherent defect in subjects of migraine which rendered them sensitive to the various exciting influences. There seemed to be a suppression of the occipital cortex with substitution or liberation of the lower and less coördinated functions.

Dr. Spriggs thought that too much might be made of the distinction between symptomatic and idiopathic migraine. Out of 100 cases of headache analyzed and investigated by him only 12 cases could be considered definitely migraine, whereas there were 36 cases in which the headaches were migrainous in type but were found to be symptomatic.

Dr. Potts said that it was suggested that migraine, starting in adolescence, as was normal, was a reaction to the fear of the responsibility of life. It had been shown that in middle life the incidence of psychosis was greater in the unmarried, possibly because they were not taking upon themselves the full responsibilities of life.

Dr. Nesbit as a personal sufferer considered heredity a doubtful etiological factor. The two theories which appealed to him most were (1) the vasomotor disturbance, (2) the pituitary enlargement.

Dr. Worster Drought thought that migraine and epilepsy were very closely allied. He had seen a case in which petit-mal in childhood had been replaced by migraine in later life. He had nearly always found a refractive error in migrainous cases, but correction of the error often afforded no improvement. He considered luminal the most effective drug for treatment.

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. H. DICKINSON, London.

An ordinary meeting of the Section took place on Friday, November 12th, when the chair was occupied by the new President, Mr. Ernest Clarke, C.V.O.

Case of Choroiditis at the Macula.

Mr. Cole Marshall exhibited a case of this character not only because of the ocular appearances, but for its clinical interest. In 1924 the patient came to hospital complaining of trouble in the right eye, the first indications of which were that the needle she was sewing with appeared to be bent, when it was not. Two or three days later she perceived two spots in the right visual field. Examination of the patient in a general way revealed pus in the right antrum, but at the time she could not be admitted as an in-patient. Vision fell from $\frac{6}{6}$ with glasses to less than $\frac{6}{60}$ in less than a month. An operation was then done on the right maxillary antrum. There was acute hemorrhage, and the eye condition seemed to be upset by it. Eventually it quieted down, and remained quiet 18 months, and then she returned with the complaint that she saw flashes in the left eye. Two small patches of choroiditis were found in the left macula, and he sent the patient to Mr. French, who washed out the antrum, which was found to be filled with pus. Thereafter the condition cleared up.

Mr. Rayner Batten said the condition of septic macula was typical at the early stages, later the appearance was not so

typical. He showed pictures of macular conditions somewhat similar to those in Mr. Cole Marshall's case.

Sir William Lister referred to the case of an officer who, during the War, was shot through the thigh, and for four years thereafter he had a discharging sinus in the femur. No complaint with regard to the sight was made until an enterprising surgeon decided on excising the sinus and scraping the bone, and this he did and sewed the wound up completely. Four days after this, the patient began to complain of failure of sight. Sir William, on being called to the case, suggested to the surgeon that he had placed a cork in the bottle, and he saw fine changes in both retinæ, at the macula. The surgeon declined to re-open the wound, but three weeks later it opened spontaneously, and fair sight was thereafter recovered, *i.e.*, from $\frac{6}{24}$ to $\frac{6}{6}$ in one eye, $\frac{6}{12}$ to $\frac{6}{6}$ in the other.

Mr. Malcolm L. Hepburn opened a discussion on

Coloboma at the Macula.

He explained that coloboma really meant mutilation, a term which could be held to include many diseases and abnormalities of structure at the macula. But the macula was a region, and there could not be a coloboma of a region, there must be a defect of some particular structure. It was properly applicable to gaps in anatomical structures, those in this region being in most cases due to some congenital mal-development. The method of formation he must leave to be discussed by embryologists, and he could not claim to be such. He therefore discussed the differential diagnosis of various conditions which were found reported under the term coloboma of the choroid at the macula. Only a small amount of material was available for examination, but on the universally accepted facts, he contended that a much more accurate diagnosis than was usual could be made. To speak of degenerations and atrophic patches, without any qualification, was almost offering an insult to the ophthalmic pathologist and his work. And it was most important to strive to the utmost to arrive at a right conclusion in regard to the various diseases and scars of the choroid, as on this depended the advice to be given to the patient. When the vision, especially central vision,

was interfered with, two or three questions were usually asked: Was it likely to affect the other eye? Was a recurrence probable? Would the present defect remain permanently? These could not be properly answered, nor a satisfactory treatment adopted, unless the fundus appearances were correctly interpreted. For example, if the condition was inflammatory, quite possibly the other eye would become involved, and that an acute choroidal deposit might occur in the same eye, either at a fresh site or in the situation of the old scar. A line of treatment must therefore be entered upon which had as its object the elimination of all sources of infection, and the prevention of further metastatic deposits. But if the condition was a true coloboma, patients could be reassured as the other eye would not be involved, though the defective vision in the affected eye could not be restored. Such was not a case for any form of treatment. If the condition was a disease of blood vessels which interfered with the due nutrition of the choroid in any fundal region toxic causes need not be sought, but attention should be directed to the vascular system, in the hope that the nutrition and function of the affected area could be restored. Mr. Hepburn passed to a description of an admitted coloboma in which there was a typical gap in the choroid in the lower part of the fundus, accompanied by a coloboma of the iris in the same situation. There was found to be a large pearly-white area with a well-defined border, edged by a narrow band of pigment, which showed where the choroid ceased. No pigment proliferation could anywhere be seen, and the only vessels crossing the gap were one or two atypical ones. Any supposed coloboma of the choroid must, to deserve the name, conform with this description. The paper was illustrated by a series of drawings, shown by the epidiascope.

Mr. Rayner Batten showed a series of drawings of the condition also, and demonstrated them on the screen.

Miss Ida C. Mann entered upon an exhaustive examination of the various views which had been advanced at different times to explain the occurrence of these colobomata. She said there were three main types: (1) those with heaped up pigment. These were characterized by a round or oval patch in the central region, and often retinal vessels ran over the

patch, showing that it was deep to the surface. Large choroidal vessels could sometimes be seen under the pigment. The chorio-capillaris layer was absent. (2) This showed punched-out holes, with a narrow ring of pigment with a pearly-white base. The base of the patch might be ectatic. (3) the type associated with some abnormal vessels.

Miss Mann said the various theories which might be advanced could be arranged under five heads. The first was that it was a true coloboma, due to a developmental anomaly in the growth of the optic cup. The second class held that it was due to an anomaly of differentiation of certain cells. The third was that it was caused by intrauterine inflammation of the choroid. The fourth theory was that it was a result of interpartum retinal or choroidal hemorrhage, and fifthly, it was held that in some of the cases the cause was an inflammatory condition occurring in early infancy. Each of these she examined at length, leaning herself to the third, and she proceeded to sketch the development of the eye in other mammals.

Mr. R. C. DAVENPORT exhibited two patients from his clinic under Mr. J. H. Fisher, who were typical of two of the types which Miss Mann had referred to.

SIR WILLIAM LISTER showed the pictures of a case which strongly suggested that the condition was of inflammatory origin. The disc was swollen and the edge blurred, while white lines ran along the course of the vessels. Both eyes were affected.

Mr. TREACHER COLLINS, in a number of instructive slides, traced the sequential stages in the development of the eye, and commented on the various views which had been advanced.

THE PRESIDENT showed pictures of and described a case which he had watched thirty years. As a baby, that patient had coloboma at the macula, in both eyes. He had passed through Harrow School, and had taken his degree at Cambridge, and had been able to participate in sport. But he did not look at one directly. This young man had had several defects, such as undescended testes and a defective facial bone, as well as horizontal nystagmus as a baby.

Mr. AFFLECK GREEVES said he had always regarded coloboma at the macula as of inflammatory origin; the heaping-up

of pigment was difficult of explanation on any other ground. The defect was obviously one of the mesoblast, not in the optic cup. In the pre-natal cases there was generally some ectasia, while in the post-natal ones the areas were flat.

Mr. HARRISON BUTLER described a case of his own, and Mr. Hepburn, in a brief reply, urged the adoption of a more exact nomenclature.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY.

Abstracts by DRS. A. N. ALLING, New Haven; P. G. DOYNE, London; and. K. WESSELY (*Archiv für Augenheilkunde*), Würzburg.

Edited by MATTHIAS LANCKTON FOSTER, M.D., F.A.C.S., New Rochelle, N. Y.

(*Continued from January issue.*)

VI.—THE MOTOR APPARATUS OF THE EYE.

97. BICHEL. **Ocular troubles following lesions of the medulla oblongata.** *Revue générale d'Ophtalmologie*, March, 1923.

98. CAPRA. **Syndrome of Marcus Gunn.** *Bolletino d'oculistica*, May, 1922.

99. CARMICHAEL and CRITCHLEY. **The relations between eye movements and other cranial muscles.** *British Journal of Ophthalmology*, February, 1925.

99a. HOFFMAN, W. F. **Paresis of convergence following encephalitis lethargica.** *Amer. Jour. of Ophth.*, ix., 823, 1926.

100. HOHMAN, L. B. **Forced conjugate upward movements of the eyes.** *Journal A. M. A.*, May 16, 1925.

101. KESTENBAUM. **Inverted Bell's phenomenon consequent upon lagophthalmos.** *Zeitschrift f. Augenheilkunde*, li., p. 367.

102. LOEHLEIN. **Retraction movements of the human eye.** *Med. Verein Greifswald*, December, 1922.

102a. LOWELL, JAY WEBB. **Surgical treatment of squint.** ARCH. OF OPHTH., ix., 600, 1926.

103. MERLE and FROGÉ. **Transitory diplopia following lumbar puncture.** *Annales d'oculistique*, clx., 1, p. 24.

104. MEYERHOF, M. **Paralyses of the ocular muscles occurring during grippe.** *Zeitschrift f. Augenheilkunde*, xlix., p. 208.

105. O'BRIEN, C. S. **Ocular muscle balance following prolonged monocular occlusion.** *Jour. A. M. A.*, lxxxv., p. 1295.

105a. PERKINS, ORMAN C. **Vertical nystagmus with fixation phenomena.** *Amer. Jour. of Ophth.*, ix., 588, 1926.

105b. PETER, LUTHER C. **Contributing factors in failure to correct convergent squint.** *Amer. Jour. of Ophth.*, ix., 253, 1926.

106. SZASZ, T. and RICHTER, H. **Otogenous abscess of the cerebellum and paralysis of the trochlearis.** *Klin. Wochenschrift*, xi., p. 501.

BICHEL (97, **Ocular troubles following lesions of the medulla oblongata**) describes a case in which, in addition to other nervous troubles, diplopia was present only when the eyes were in the position of rest. The patient was a man 42 years old, with no special history, who was seized suddenly with pain in the right side of the back of the head and attacks of dizziness which caused him to fall without losing consciousness. The symptoms which affected the nervous system only of the otherwise healthy man, may be divided into four syndromes, pertaining to the brain, the sensibility, the cranial nerves, and the sympathetic. The patient staggered and had a tendency to fall to the right, yet Romberg was negative. The brain symptoms were manifested through hypotony, asynergy, and catalepsy of the entire anterior half of the body; nystagmus on looking to the right, a weaker nystagmus on looking to the left, hemianæsthesia of the type of syringomyelia. Almost exactly vertical double images appeared only when the eyeballs were in the position of rest, they disappeared as soon as the eyes were moved. A slight involvement of the trigeminus was shown by a reduction of the corneal and conjunctival reflexes. The left corner of the mouth hung down. Hearing was intact. There was difficulty in swallowing and hoarseness with paralysis of the right side of the soft palate and of the right vocal cord. There was miosis of the right eye, the pupils reacting to light and convergence. Normal fluid was obtained by a lumbar puncture; Wassermann was negative with both blood and cerebrospinal fluid. All of these symptoms, especially the diplopia, indicated a lesion in the right side of the medulla oblongata just back of the olive. The author ascribes the peculiar form of diplopia to the sympathetic, which plays an important part in the muscle tone during the condition of rest.

SZASZ and RICHTER (106, **Otogenous abscess of the cerebellum and paralysis of the trochlearis**) claim that this is the first case of the sort to be recorded. The autopsy revealed pressure upon the nerve at the place where it passes between the mesencephalon and the anterior median portion of the hemisphere to the base of the brain.

MEYERHOF (104, **Paralyses of the ocular muscles occurring during grippe**) observed twenty-eight cases of paralysis of ocu-

lar muscles during an epidemic of grippe and encephalitis during the past year. The abducens was affected in eleven cases, the oculomotor in four, the trochlearis in one, the accommodation of both eyes in four, and combinations of these in the rest. Almost all recovered in from one to eight weeks. He noticed in some cases a direct transition of grippe encephalitis with paralyses of the ocular muscles into encephalitis lethargica, and was unable to differentiate between the two diseases. He thinks that in rare cases a peripheral toxic neuritis may be assumed to be the cause of the ocular paralyses in grippe, and that occasionally inflammation of the accessory nasal sinuses may be the cause.

HOFFMAN (99a, **Paresis of convergence following encephalitis lethargica**) reports seven cases of convergence weakness coming on after the active manifestations of the disease had disappeared. No ocular muscle seemed to be involved in any of the cases. He was unable to locate the exact area of the lesion. YUDKIN.

CARMICHAEL and CRITCHLEY (99, **The relations between eye movements and other cranial muscles**) say that the best known example of associated movement is the jaw-winking reflex, in which movement of the jaw is associated with movement of the lid. In this paper six other types of associated movement are described.

(1) The oculo-aural movement. If the eyes be fully deviated laterally, the helix of each pinna becomes rotated backwards and inwards towards the mastoid. It is a bilateral phenomenon, the wider excursion occurring in the contralateral ear.

(2) The oculo-frontalis movement. On deviating the eyes laterally there is a depression of the contralateral eyebrow and a raising of the homolateral eyebrow.

(3) The oculo-lingual movement. The point of the tongue may turn over to the homolateral side in deviation of the eyes.

(4) The oculo-mandibular movement. If the eyes be rapidly deviated from side to side, a protrusion and deviation of the mandible to the contralateral side occurs.

(5) The orbiculo-stapedial movement. This consists in the occurrence of a buzzing noise in the ear associated with a homolateral blepharospasm.

(6) The oculo-nasal movement. On extreme deviation of the eyes, a dilatation of the nostrils occurs.

The authors discuss the ætiology of these movements and favor the view that they are associated movements, vestigial in origin, and relics of purposive and more specialized movements.

HOHMAN (100, **Forced conjugate upward movements of the eyes**) reports four cases of involuntary turning up of the eyeballs and a holding of the eyes in that position. The patients are unable to turn the eyes down, and they are unaware of any pulling or tension. The spasm is not painful and is only temporary. These patients show other symptoms of post-encephalitic Parkinson's syndrome.

ALLING.

CAPRA (98, **Syndrome of Marcus Gunn**) describes three cases. The nature of this anomaly is that in some eyes with ptosis movements of the lower jaw cause the upper lid to be raised simultaneously. The ptosis is almost always confined to the left eye and is congenital. Frequently the eye is also amblyopic and one or more of the extrinsic muscles are paretic. The question is evidently one of a stimulation from the innervation of the lower jaw which is transmitted through central or peripheral anastomoses to the innervation of the levator. As yet nothing certain has been determined concerning the way in which this transmission takes place. The use of the galvanic current has proved of some value therapeutically.

KESTENBAUM (101, **Inverted Bell's phenomenon consequent upon lagophthalmos**) reports the case of a woman, 30 years old, whose right upper lid was drawn up by a deep scar. The upper third of the cornea was badly affected by a keratitise lagophthalmo, and the upper part of the conjunctiva also was much changed. On closure of the lids the right eye made abnormal movements which he designated as an inverted Bell's phenomenon. On gentle closure the lower lid rose high, while the upper lid was retracted still further and, at the same time, the eyeball turned down leaving about *2mm* of the upper part of the cornea uncovered. At the same time the healthy left eye moved somewhat upward. On strong closure of the lids the lower lid rose still higher, so that only

the temporal portion of the palpebral fissure remained open, and the eyeball turned down still further so that it was well covered. At the same time the sound eye rose still higher. On changing to the gentle closure the left lower lid sank and the eyeball turned down still further so that it was well covered. At the same time the sound eye rose still higher. On changing to the gentle closure the left lower lid sank and the eye again turned somewhat downward. Kestenbaum is led to think by these very dissociated movements of the two eyes that Bell's phenomenon is a movement of expediency and not dependent on a congenital firm connection between the movements of the lid and of the globe.

HOEHLEIN (102, **Retraction movements of the human eye**) points out that in all the described cases abduction was almost completely absent and retraction accompanied attempts at adduction. The usually accepted theory of a transformation of the external rectus into a fibrous band furnishes a better explanation than that of an abnormally situated retractor of the eyeball. It is also supported by the cases reported by Dippelt and Loehlein, in which an exactly similar picture was produced by a cicatricial fixation of the externus. In congenital retraction movements the trouble is probably due not to a primary defect but rather to pressure with a consequent degeneration of the muscle.

MERLE and FROGE (103, **Transitory diplopia following lumbar puncture**) observed a case of paralysis of both externi six days after a lumbar puncture in a woman 42 years old. Spontaneous recovery took place within a week. The theory that this was of hysterical origin was set aside as untenable. Paralyses of ocular muscles have been observed after lumbar anæsthesia. In this case two hypotheses present themselves; it was either a paralysis of the two externi of preatactic nature made manifest transiently by the lumbar puncture, or it was a functional diplopia caused by vasomotor disturbances, such as are met with in ophthalmic migraine. Time alone can tell which, if either, of the two hypotheses is the correct one.

O'BRIEN (105, **Ocular muscle balance following prolonged monocular occlusion**) finds that the long method of Marlow and a shorter one which he uses is a great aid in uncovering latent ocular unbalances. This test gives a basis on which to treat

cases of extramuscular imbalance more or less similar to the basis for the treatment of refractive errors following cycloplegic. By this method of occlusion the eye is allowed to seek its relative anatomic position of rest. ALLING.

With the aid of special instruments and locking tendon forceps, LOWELL (102a, **Surgical treatment of squint**) is able to secure a definite amount of tucking or recession of a muscle. The muscular imbalance is determined by a specially constructed instrument which resembles the tangent scale tropometer originated by Nicati and improved by Stevens.

YUDKIN.

PETER (105b, **Contributing factors in failure to correct convergent squint**) finds that failure to secure binocular vision after an operative procedure is due to either a refractive error or the presence of amblyopia exanopsia or defective fusion faculty. A cosmetic failure is usually due to a poor choice of operation or defective surgical technic. YUDKIN.

PERKINS (105a, **Vertical nystagmus with fixation phenomena**) describes a vertical nystagmus accompanied by an upward fixation of the eye which remains in this position regardless of the patient's attempts to bring them down. This ocular syndrome is observed as a sequela following encephalitis lethargica and is probably due to an organic disturbance.

YUDKIN.

VII.—INTRAOCULAR TENSION AND GLAUCOMA.

107. AMSLER. **Tonometric observations.** *Revue Générale d'Ophtalmologie*, September, 1922.

108. BAILLIART. **The retinal circulation with intracranial hypertension.** *Annales d'oculistique*, clix., 11, p. 785.

109. BESELIN. **Some experiences in operations for glaucoma, especially cyclodialysis.** *Klin. Monatsbl. f. Augenheilkunde*, May-June, 1923, p. 761.

110. BONNEFON. **The action of adrenalin in glaucoma. Hypertension from vasoconstriction.** *Annales d'oculistique*, clx., 6, p. 470.

111. CALHOUN, F. P. **The non-operative and operative treatment of glaucoma.** *American Journal of Ophthalmology*, November, 1925, p. 849.

112. CORDS, RICHARD. **Cyclodialysis the best operation for simple glaucoma.** *Ibid.*, May, 1924, p. 341.

112a. DAVENPORT, R. C. **After results of corneo-scleral trephining for glaucoma.** *British Journal of Ophthalmology.* Sept., 1926.

113. ELIASBERG, M. **Coincident operation for glaucoma and senile**

cataract in a highly myopic eye. *Klin. Monatsbl. f. Augenheilkunde*, April, 1923, p. 532.

114. ELSCHNIG. **Curran's iridotomy for glaucoma.** *Ibid.*, May-June, 1923, p. 667.

115. FEILCHENFELD, W. **Glaucoma in the course of an uncomplicated senile cataract.** *Ibid.*, April, 1923, p. 529.

116. FROMAGET. **Treatment of acute glaucoma by the retrobulbar injection of novocain-adrenalin.** *Annales d'oculistique*, clx., 6, p. 438.

117. GIFFORD, H. **Further experience with peripheral iridotomy (Curran) and subconjunctival limbus puncture in the treatment of glaucoma.** *American Journal of Ophthalmology*, May, 1924, p. 346.

118. v. GROSZ. **Cyclodialysis for secondary glaucoma due to subluxation of the lens.** *Ungar. Ophthalm.*, May 20, 1923.

119. MAGGIORE. **Glaucoma a late complication in idiopathic detachment of the retina.** *Annali di Ottalmolog. e Clinica oculistica*, January, 1923.

120. MAGITOT and BAILLIART. **The circulatory régime of glaucoma.** *American Journal of Ophthalmology*, October, 1925, p. 761.

121. MAGITOT, A. R. **Effect of collyria upon the hypertonic reaction of the human eye after corneal puncture.** *Annales d'oculistique*, clx., 3, p. 165.

122. NEUHAEUSER, A. **Glaucomatous increase of tension without excavation of the papilla.** *Archiv f. Augenheilkunde*, 1923, xcii., p. 235.

123. RAEDER. **Studies concerning the position and thickness of the lens in the human eye in physiological and pathological conditions. The position of the lens in glaucoma.** *Archiv f. Ophthalmologie*, cxii., p. 29.

124. REBUCCI. **Glaucoma in the province of Parma during the years 1901-1920.** *Bolletino d'oculistica*, April, 1923.

125. REITSCH. **Recurrent hemorrhage into the anterior chamber after cyclodialysis.** *Klin. Monatsbl. f. Augenheilkunde*, March, 1923, p. 401.

126. RING, G. ORAM. **Adrenalin chloride with special reference to its subconjunctival injection.** *American Journal of Ophthalmology*, July, 1925, p. 553.

127. SALUS, R. **Glaucoma and vascular nævus.** *Klin. Monatsbl. f. Augenheilkunde*, April, 1923, p. 305.

128. THIEL, R. **Studies of the aqueous and of the cerebrospinal fluid in diseases of the central nervous system.** *Klinische Wochenschrift*, 1923, No. 48.

AMSLER (107, **Tonometric observations**) found in an arteriosclerotic patient with very high blood pressure and a hemorrhagic retinitis in his right eye that the tension of this eye was lower than that of the left. Repeated measurements during treatment in the hospital showed that as the systolic and diastolic blood pressure gradually diminished the tension of

the healthy eye slightly decreased while that of the diseased eye increased. Fifteen months later the patient reappeared. At this time there were multiple retinal hemorrhages in the left eye, while the right was perfectly normal, and the tension of the left eye was distinctly lower than that of the right. The left eye was myopic.

MAGITOT (121, **Effect of collyria upon the hypertonic reaction of the human eye after corneal puncture**) says that although the effect of various collyria upon the pupil is partly known, we know very little concerning the influence exerted upon the mechanism of the eye tension. The tension of the normal eye is changed only by collyria containing adrenalin, the effect of eserine is questionable, cocaine acts only in solutions stronger than 2 per cent. Magitot comes to the following conclusions; Atropine, philocarpine, and adrenalin prolong the duration of the reduced tension of the eye after puncture of the anterior chamber. Atropine and pilocarpine hinder the hypertonic reaction, yet allow the tension to rise to at least its normal height. Adrenalin hinders that latter as well. Its effect is to be compared with that produced by ligation of the carotid of the same side Pilocarpine acts like atropine, only the reactions follow somewhat more quickly.

BAILLIART (108, **The retinal circulation with intracranial hypertension**) says that little or no notice is generally paid to the condition of the retinal vessels in choked disk, yet a slight dilatation of the veins or a thinning of the arteries may be of great importance. In the majority of cases one finds an abnormal elevation of the local diastolic arterial pressure, while the venous pressure is normal and corresponds to the intraocular. More interesting are his observations of the retinal vessels when there is no choked disk and the circulation is apparently undisturbed as frequently occurs in serous meningitis. In most cases the pressure in the retinal arteries is raised much above the normal, caused, Bailliart thinks, by a disturbance in the outflow of the blood. Fluctuations in the intracranial pressure are generally parallelled by the same in the retinal arteries. The increase of blood pressure is looked upon as a preventive measure to guard the circulation of the blood in the capillaries against the effect of the increased intracranial pressure. Usually the increased blood pressure in the

cerebral arteries goes hand in hand with an increase of the general blood pressure, which has a similar relation to the cerebrospinal fluid. By attention to this new sign in the fundus we have a means by which to diminish the number of negative conditions in elevation of the intracranial pressure.

THIEL (128, **Studies of the aqueous and of the cerebrospinal fluid in diseases of the central nervous system**) found an increased passage of fluorescin into the aqueous and at the same time a higher content of fluorescin in the cerebrospinal fluid in all the cases examined of tabes, taboparalysis, lethargic encephalitis and retrobulbar neuritis, while the aqueous contained no fluorescin in dementia præcox, tumors of the brain and meningitis, oculomotor paralysis, cerebral syphilis, and choked disk. He thinks that in the first set of cases there are changes in the epithelium of the ciliary body and the choroidal plexus which are not now known, and concerning the clinical importance of which nothing can as yet be said but which may possibly prove to be of diagnostic value.

MAGITOT and BAILLIART (120, **The circulatory régime of glaucoma**) basing their conclusions on experimental and clinical practice find that normally the arterial pressure in the choroid and retina is practically the same, the diastolic being about 30*mm* Hg and the systolic about 70*mm*. The same analogy exists for the venous systems of the choroid and retina and that the various pressures on leaving the eyeball are in equilibrium with the ocular tension. In glaucomatous hypertension these conditions are profoundly modified and the vascular régime is characterized by a considerable elevation of the venous pressures in the retinal and choroidal systems. They look upon the venous hypertension as being produced by phlebitic lesions, chiefly in the veins of the ciliary circle. This however, will not give the rise in the ocular tension of glaucoma if the capillary network functions normally. This network forms a restraining barrier, which absorbs the arterial column and transforms its strong pressure into a weak venous pressure. A disturbance of this intervening system, by some active vasodilation caused either by a nervous influence or a drug, can force the dam formed by the ocular capillaries between the arterial and venous systems and should any ob-

stacle to the return circulation exist, the ocular tension tends to increase and assumes the picture of hypertony of glaucoma.

ALLING.

RING (126, **Adrenalin chloride with special reference to its subconjunctival injections**) reports four cases of acute congestive glaucoma which were controlled by these injections. A preliminary reduction of ocular tension was noted and the gravity of the situation was lessened. It is thus possible to postpone further operation until the eye is quiet enough for surgical intervention. ALLING.

FROMAGET (116, **Treatment of acute glaucoma by the retrobulbar injection of novocain-adrenalin**) reports three cases in which he injected 3 or 4*ccm* of a 2–4% solution and obtained remarkable diminution of tension within a few minutes. In one patient, who had several attacks, the effect was demonstrated to be directly proportional to the quantity of adrenalin. When two drops of a 1–1000 solution of adrenalin was added to a *ccm* of novocain the tension sank in five minutes from 70 to 30*mm* Hg; after another five minutes to 5*mm*, where it remained for ten minutes. Then there was a slow rise of tension, in forty minutes to 30*mm*, and after some hours to its original height. In one case the tension grew less and finally disappeared, so a cure was effected without operation.

BONNEFON (110, **The action of adrenalin in glaucoma**) combats Fromaget's conclusion that the circulation of the blood in the choroid rules the tension of the eye. In certain forms of vascular glaucoma adrenalin unquestionably causes a lowering of tension when injected retrobulbarly, but in chronic glaucoma it is ineffective and in some cases causes an increase of tension. From the study of a case of chronic glaucoma he is inclined to think that the tension lowering effect of adrenalin does not depend on the condition of vasoconstriction of the intraocular vascular network, because this alone, without previous vasoconstriction of the extraocular arterial trunks generally has no influence on the tension. The iris of the patient was ischæmic after the retrobulbar injection, the choroid probably was in the same condition, and the eye was harder. Apparently the adrenalin had induced a hypersecretion of the intraocular fluids through stimulation of the sympathetic. To explain how adrenalin injected behind the

globe markedly reduces the tension in hemorrhagic glaucoma he suggests that the pathological increase of tension may be reduced by the vasoconstriction of the vessels at their entrance into the globe if the intraocular vessels are filled to the point of bursting. The fact that adrenalin used in the same way in chronic absolute glaucoma increases the tension considerably he explains as follows: As, on the one hand, the intraocular venous pressure is lower than the intraocular pressure itself, but, on the other hand, is higher than the arterial diastolic pressure, and as further the dynamic diminution of the systole, which drives the blood into the eye, permits the escape of the venous blood, a vasoconstriction just outside ot or within the eyeball may interfere with this process and induce a venous stasis with a consequent increase in tension. Instillation of adrenalin into the conjunctival sac has practically no tension reducing effect on the normal human eye, and no essential change in the tension was observed in chronic glaucoma, with the exception of one case in which the tension rose from 80 to 90 within two minutes. Bonnefon gives the following results of his experiments on rabbits: (a) Instillations and subconjunctival injections produce at first a slight, short increase of tension, then a slight hypotony, with finally a return to the original tension. (b) Retrobulbar injections of novocain-adrenalin induced almost immediately a lowering of tension that lasted several hours. (c) Injection into the vitreous produced no effect. (d) Injection into the anterior chamber after previous escape of the aqueous caused a collapse which lasted several hours. The normal tone was not restored until after twenty-four hours. A constant phenomenon in these experiments is the vasoconstriction, but the lowering of tension seems to be less dependent on this than on the ischæmia of the extraocular ciliary vessels. Bonnefon thinks that the tension of the eye is the resultant of two balanced antagonistic forces, the parietal tone of vascular origin and the visceral tone dependent on the filling of the globe with fluid. Hence he distinguishes a parietal hypertony caused by a sudden overfilling of the choroid, which may give rise to ruptures of the vessels (hemorrhagic glaucoma), or acute œdema ot the vitreous (acute glaucoma), and a visceral hypertony caused by an accumulation of the intraocular fluids, which leads secondarily

to functional and trophic disturbances through an advancing under pressure of the parietal circulation. From this conclusions may be drawn concerning the indications and contra-indications to the use of adrenalin in glaucoma. It is indicated in the parietal forms of glaucoma, contraindicated in the visceral. Yet Bonnefon reports a case of acute glaucoma in which repeated retrobulbar injections of novocain-adrenalin produced not the slightest result.

CALHOUN (111, **The non-operative and operative treatment of glaucoma**) reviews the subject by dividing his cases into congestive and non-congestive types. The congestive group except in unusual cases should be operated on. He prefers general anæsthesia to local in order that the personal equation of the patient does not enter into performance of a perfect operation. Medical treatment does not seem to give much satisfaction in this condition. In a small proportion of private patients having the non-congestive type of glaucoma medicinal treatment should be tried for a reasonable time, carefully watching the vision, visual fields and tension. Should there be progressive reduction in the first two after the use of miotics, and treatment for evidenced disorders having a bearing upon increased intraocular tension, an operation should be undertaken. He finds that some form of a filtering cicatrix is better in this type than just merely an iridectomy.

ALLING.

BESELIN (109, **Some experiences in operation for glaucoma especially cyclodialysis**) recommends this operation, particularly for glaucoma simplex. Of forty eyes operated on he kept thirty-six under observation for from six months to six years and obtained a permanently good result in fifty-five per cent. In order to avoid movements of the eye during the incision he fixes the eye with two forceps, one on each side of the cornea horizontally. Among the advantages of cyclodialysis over iridectomy in simple glaucoma he mentions the absence of wound astigmatism and coloboma. He has seen one late infection after trephining. Sometimes anterior sclerotomy gives a good result.

CORDS (112, **Cyclodialysis the best operation for simple glaucoma**) is of the opinion that in glaucoma simplex the operative treatment should always be commenced with this

operation. It is the easiest bulbar operation and the possible complications are very few. The influence upon the tension is on the whole very satisfactory and may be readily repeated if the result is not sufficient. In refractory cases, any other operation may be made afterwards. ALLING.

v. GROSZ (118, **Cyclodialysis for secondary glaucoma due to subluxation of the lens**) employed this operation in five cases. In four the tension was reduced to normal, in the fifth it was at least lowered somewhat. This observation is the more important as the usual operations for glaucoma are so apt to give bad results in this disease, and are attended by so many dangers.

REITSCH (125, **Recurrent hemorrhage into the anterior chamber after cyclodialysis**) reports the case of a woman 76 years old, who had a high degree of arteriosclerosis, thrombosis of a retinal vein, and chronic glaucoma in both eyes. On the day after a cyclodialysis a hemorrhage appeared in the anterior chamber, accompanied by a slight pain. The hemorrhages recurred nine times and ceased after homatropine had been administered for three days.

ELSCHNIG (114, **Curran's iridotomy for glaucoma**) has tested Curran's operation on sixteen eyes and recommends it for both acute and chronic cases in which the anterior chamber is shallow. He considers it to be a palliative measure in cases ot acute glaucoma with abolition of the anterior chamber.

GIFFORD (117, **Further experience with peripheral iridotomy (Curran) and subconjunctival limbus puncture in the treatment of glaucoma**) found that iridotomy did by no means give the desired result in all cases and was not to be classed with practically harmless ones. He substitutes a subconjunctival limbus puncture as a preliminary operation to relieve tension. ALLING.

ELIASBERG (113, **Coincident operation for glaucoma and senile cataract in a highly myopic eye**) observed an acute attack of glaucoma in a cataractous eye of a woman 74 years old, whose other eye had been operated on previously for cataract. The eye which had been operated on was emmetropic for distance and presented ophthalmoscopically a large posterior staphyloma. An iridectomy was performed and the

cataract extracted without accident. After recovery the fundus and the refraction were found to be like those of the other eye.

FEILCHENFELD (115, **Glaucoma in the course of an uncomplicated senile cataract**) reports what was unquestionably a case of an attack of glaucoma induced by a rapidly swelling cataract. The patient, who was known through a former examination to have a nuclear cataract in each eye, was seized suddenly with severe pain in the head and right eye and vomiting. The eye was not reddened, the cornea was clear, the anterior chamber very shallow, the lens much swollen, the pupil dilated and irresponsive to light, the tension very high. Extraction with iridectomy was performed with good result. The patient had a high degree of arteriosclerosis.

MAGGIORE (119, **Glaucoma a late complication in idiopathic detachment of the retina**) maintains that many eyes with detachment of the retina develop sooner or later a painless, plastic iritis which leads to secondary glaucoma. He describes four cases with the histological findings. In three cases the detachment was due to myopia, in the fourth the cause was unknown. All presented extensive uveitis, seclusion, and the characteristic marks of a secondary glaucoma with its sequelæ,—bullous keratitis, complicated cataract, and optic atrophy. In no case was the ciliary body involved in the inflammation, but the unimpaired secretion of this seems to have led to the increase of tension.

This paper, by DAVENPORT (112a, **After results of corneoscleral trephining for glaucoma**) reviews the results of corneoscleral trephine operations for glaucoma performed at Moorfield's Eye Hospital during the years 1919–1923. 636 records were investigated. Of these 100 were cases of obviously secondary glaucoma and were not considered further. For purposes of comparison the cases were divided into three groups, A vision 6/6–6/12, B vision 6/18–6/60, C less than 6/60. Certain cases in group C after operation improved into group A. These cases were mostly those of acute glaucoma in which the initial poor vision was due to circulatory disturbance and hazy media rather than damage of nerve fibers. With the exclusion of these there are left 384 cases. These 384 cases before operation lay grouped as follows:

Group A (6/6–6/12).....154
Group B (6/18–6/60).....104
Group C (less 6/60).....127

and after operation:

Group A.....155
Group B.....106
Group C.....124

In judging the effects of the operations, very varying records had to be relied upon. Perimetric records were apt to be irregular and the attendance of patients, especially of those in whom apparently the greatest success was attained, was often infrequent. It was found that the comparison of the visual records under this grouping gave the most useful information. It would appear from the foregoing figures that the visual acuity is not greatly altered for better or for worse by the trephine operation, and that the mutilation required by the operation is minimal, and that in all probability in the majority of cases the normal tension was maintained by means of the operation as the visual results show no gross deterioration and at least three years have passed since operation. In group A (vision 6/6–6/12) the cases are of the type most favorable for operation and of these cases 80% were successes and only 4.5% complete failures. The operation in use was the typical Elliot operation with the 1.5*mm* trephine, the only modifications occasionally employed being complete iridectomy instead of peripheral buttonhole and careful suturing of the flap in its whole length.

Complications.

Vitreous loss: 7 cases reported, all in old patients, 3 eyes had to be excised.

Loss of disk in the eye: Noted in 3 cases, no ill effects produced.

Hemorrhage: 8 patients noted as having hemorrhages in the fundus after operation.

Iris prolapse: 2 cases noted, in neither case was iridectomy

done at operation. One did well, the other late infection occurred.

Detachment of choroid recorded in 10 cases and in all the detachment subsided rapidly, and had apparently no ill effect.

Infection: (1) Quiet iritis seemed to be the rule after the operation; in spite of atropine instilled after operation. It appeared that this quiet iritis had no ill effects as regards resulting vision or tension.

(2) Acute infection was recorded in 2 cases following immediately after the operation.

(3) Late infection. 14 cases are reported. Most occurred within a few months of the operation; one case, however, is reported occurring 7 years after operation. Evisceration was necessary in 2 cases, and in 6 of the cases the inflammation subsided, leaving vision from 6/12− to 6/18−.

Relapse of Tension: In a few cases operation had to be repeated. This was generally in old patients. There appears from these records that there is a certain type of case, generally in old people, in which the tension cannot be kept down by operation, repeated operations of varying types, trephining, sclerotomy and iridectomy all failing in their object. Also, that there is a malignant type in all ages hard to check and control, and also a type with normal or low tension with, in other respects, progressive glaucoma changes in which operative measures fail.

Cataract: There was little evidence that trephining predisposed to cataract, except in unsuccessful or imperfect operations when cataract was likely to follow. The results of extraction of lens after a previous trephining operation showed success technically, though often disappointing results visually.

DOYNE.

That there is danger of glaucoma in an eye near a vascular nævus has been shown by nine cases heretofore reported. All of these were in children or young people, and, with one exception, buphthalmos was always present. SALUS (127, **Glaucoma and vascular nævus**) reports two cases met with in adults. The first case was that of a woman 25 years old, who consulted him on account of conjunctival trouble. She had a large vascular nævus on her left cheek and lids. The

conjunctival vessels over the lower part of the eye were much increased. The margin of the cornea was so very vascular as to have a violet color. The vascular condition was less marked above. The papilla was excavated to its lower and outer margins. Vision was perfect. The visual field showed a narrow scotoma arising from the blind spot and surrounding the field of fixation. Tension was 30*mm*. The second case was that of a man 57 years old who had a vascular nævus involving the left lids, conjunctiva and episclera. The condition was less marked above. The papilla had a distinctly sharp edged excavation and was gray white. Vision was good. A large scotoma extended from the blind spot passing about the point of fixation above. Tension was 26*mm*: that of the other eye 16*mm*.

NEUHAEUSER (122, **Glaucomatous increase of tension without excavation of the papilla**) reports two cases. The first case was that of a woman 42 years old, whose eyes appeared normal externally and ophthalmoscopically, although the tension had been high for about a year and a half. Miotics contracted the pupil, but did not lower the tension. The second patient was 21 years old and came under treatment in the latter part of 1920. Subjectively he complained of pressure pains after prolonged night work and the seeing of clouds and rainbow colors with his left eye. Both eyes were unirritated, the left anterior chamber was rather shallow with signs of scattered pigment, the left pupil almost irresponsive to light with wormlike contractions of the sphincter, a condition present also in the right eye in which the pupillary reaction was normal. The left papilla was rather more hyperæmic than the right, but was otherwise perfectly normal. Vision of each eye normal. No scotomata. Adaptation to the dark markedly reduced in the left eye. Miotics contracted the pupil scarcely at all and had no effect on the tension. The tension was elevated, running as high as 57*mm*, except for a lowering during the warm months. It is noteworthy that the nervous tissue had suffered no harm in spite of the considerable increase of tension. The disturbance of adaptation was explained by the rigidity of the left pupil, as the right dilated to the maximum in the dark.

REBUCCI (124, **Glaucoma in the province of Parma during**

the years 1901-1920) says that glaucoma affected 1.65% of all the patients treated in the eye clinic during these years, equally divided between men and women. It occurred most frequently between the ages of 60 and 70, and was more common in the winter than in the summer in the ratio of 187:97. The condition was met with as a rare exception in myopes; the overwhelming majority of the patients were hypermetropic. The different forms were met with in the following proportions; Simple glaucoma, 7.70%; acute inflammatory, 26.36%; chronic inflammatory, 55.65%; secondary, 10.27%.

RAEDER (123, **The position of the lens in glaucoma**) first describes the topographical anatomy of the vitreous and of the diaphragm of the eye, which consists of the anterior limiting layer of the vitreous, the fibers of the zonula and the lens, with only passive involvement of the iris. The diaphragm is only slightly permeable to fluid, so it acts as an obstacle and is arched in the direction of the least fluid pressure. There is no free current of fluid between the chambers of the vitreous and the aqueous. The permeability of the diaphragm is proportional to the logarithms of the pressure difference. When a colored solution is introduced into the vitreous under pressure, a colorless fluid passes into the anterior chamber for perhaps forty-eight hours, until the permeability for fluid ceases to exist. The colored fluid enters but little into the vitreous, but presses it together from the side, and what enters the anterior chamber is vitreous fluid. By increase of the differential pressure the aqueous is driven out and the anterior chamber emptied, then the diaphragm comes into balance, and the quantity of fluid passing through corresponds to that which has been driven out. Every difference of pressure between the vitreous and the anterior chamber finds expression in a corresponding depth of the anterior chamber. When there is overpressure in the vitreous the anterior chamber becomes shallow and the diaphragm is arched away from the vitreous, but when the overpressure is in the anterior chamber this becomes deep and the diaphragm is arched toward the vitreous. The elastic tension of the diaphragm thus acts as a differential equalizer. The depth of the anterior chamber varies with the pressure in it

and in the vitreous. Clinical conditions mentioned in which the depth of the anterior chamber is of moment are: 1. Hypertension with shallow anterior chamber. In glaucoma secondary to tumor of the choroid the shallowing of the anterior chamber occurs before or simultaneously with the increase of tension. The pressure is dependent on the size of the tumor and the quantity of exudate detaching the retina. Such is the case also in glioma of the retina, panophthalmitis, and hemorrhagic glaucoma. Primary acute glaucoma always has a shallow anterior chamber which appears before the attack and is present in the other, not diseased eye. This is confirmed by measurements made by Raeder. An explanation suggested is that the iris becomes thickened by the stasis in the anterior segment so that the decrease in depth of the anterior chamber may be apparent, but it is also due to a forward movement of the diaphragm as the pressure in the vitreous is the greater. In the glaucomatous eye less fluid enters the anterior chamber because either passages for fluid at the ciliary ring are tamponed by the lens, or the power of the vitreous to take up water is greater. In seclusion of the pupil, or the secondary glaucoma induced thereby, there is only an apparent shallowing, as it is the iris alone, and not the lens which is driven forward. The accumulation is in the posterior chamber, not in the vitreous as it is in true glaucoma. 2. Hypertension without shallow anterior chamber. An example of this is the glaucoma produced experimentally by occlusion of the angle of the anterior chamber with electrolyzed iron. With the first increase of tension the anterior chamber becomes deeper. A similar condition is found in buphthalmos, in which the deep anterior chamber and the increase of tension are due to the faulty development of the outlet in the angle of the chamber. In secondary glaucoma with cyclitis the depth of the anterior chamber is normal or increased, because the pathological process lies in front of the lens, with perhaps the albuminous character of the aqueous as another cause. The same explanation applies in cases of cysts of the anterior chamber, and occlusion of the filtration network by blood cells, exudates, fibrin, lenticular fragments, or pigment. Three kinds of glaucoma may be differentiated, that in which the primary expansion is

situated in the posterior segment of the eye, that in which it is situated in the anterior segment, and third that in which it is situated in the posterior chamber. 3. Chronic primary glaucoma in which the anterior chamber may or may not be shallow. There are glaucomas with an insidious course without signs of stasis or clouding of the cornea. Usually the anterior chamber is of normal depth, sometimes it is shallow, sometimes deep. There is no simple ratio here between the tension and the depth of the anterior chamber as the highest tension may be accompanied sometimes by a shallow, sometimes by a deep anterior chamber. One may assume that there are two groups which correspond somewhat to simple and to chronic inflammatory glaucoma, which often cannot be differentiated clinically. A transition may be recognized first when chronic primary glaucoma with distinctly deep anterior chamber has an attack. No differentiation can be made through the variations in the depth of the anterior chamber, because the values vary. With a shallow anterior chamber associated with a small cornea, hypermetropia, and microphthalmos, the expansion is assumed to be in the posterior segment, with a deep anterior chamber in the latter. An attempt to classify the primary glaucomas according to the depth of the anterior chamber gives the following: A. Shallow anterior chamber. 1. acute inflammatory glaucoma. 2. Chronic glaucoma with or without inflammation. B. Normal or deep anterior chamber. 1. Infantile glaucoma (hydrophthalmos); 2. juvenile glaucoma; 3. senile glaucoma. In cases of chronic glaucoma which have been operated on a shallow anterior chamber is found with low tension because of the greater filtration from it. When the tension increases the depth of the chamber does likewise because filtration has lessened.

(*To be continued.*)

CORRESPONDENCE.

Results in Treating Trachoma Among Indians.

Editor, Archives of Ophthalmology.

Dear Sir:

Trachoma, as we see it, varies a great deal in malignancy, form and duration. Its response to treatment, both medical and surgical, also varies to an extraordinary degree. In an occasional case, due probably to extreme virulence of the infection or lack of resistance on the part of such patients, there is little or no response at all to treatment. In very recent cases and the acute exacerbations of old cases, I believe that medical treatment only should be employed. The method that we have found most satisfactory in the majority of cases has been daily applications to the trachomatous conjunctiva, by means of a cotton tipped applicator, of a one or two per cent. solution of silver nitrate, the lids being held open until a white film forms, then neutralizing the excess by irrigating the eye with boric acid solution. In the later stages of recent trachoma, when there is not much secretion, we find that the copper sulphate pencil applied lightly to the diseased conjunctiva is very useful. During the more chronic stages of the disease, the stages in which the bulk of our patients present themselves for treatment, we have found that surgical combined with medical treatment produces results in most cases in a much shorter period of time than could be obtained by medical treatment alone. And, we believe, with little or no more damage to the eye, if care is used in selecting cases and the operation be not too radical, than if medical treatment only is used. The method gener-

ally employed is mild grattage, repeated two or three times, or oftener if necessary, during the year and the daily treatment with silver solution or copper sulphate during the intervals between operations. The eye is cocainized and the lids everted over a lid elevator. After the follicles are broken up, uncapped, with a dull curette or other semi-sharp instrument the trachomatous conjunctiva is picked up between layers of gauze, wet with 1:1000 solution of bichloride, with the thumb and forefinger and thoroughly squeezed, being careful to avoid tearing the tissue, then gently rubbed. The follicular contents can thus be evacuated with a minimum of damage to the tissues. A very good method, also, is to roll out the follicles with Knapp's forceps or milk or strip them out with Princes' forceps and then evert the lid over a horn plate, or special lid plate with retractor attached which we have devised for the purpose, and gently scrub the affected conjunctiva with a gauze sponge wet with the bichloride solution.

After grattage, for four or five days or until the reaction subsides, the eyes are treated only with argyrol or other simple non-irritating collyria. In the majority of cases suitable for grattage, which in my judgment constitutes about ninety per cent. of all cases suitable for any kind of surgical treatment, we would emphasize the importance of gentleness and care on the part of the operator to attack, in so far as possible, only diseased conjunctiva thus avoiding damage or destruction of tissue that would probably not be destroyed by the disease.

We have performed the so-called radical grattage, incising the conjunctiva and scrubbing it vigorously with a stiff bristled toothbrush, but we are now convinced that much unnecessary damage is done by such harsh procedures. Symblepharon and extensive, deep cicatrization quite commonly occur after such an operation.

Experience has taught us that the mild, conservative operation, even though it takes longer to eradicate the disease, gives far better ultimate results. By conservative methods we at least do not produce conditions that are more distressing to the patient than the disease, and which would in the future reflect on our judgment as physicians and discredit our efforts.

In dealing with the old chronic cases of many years duration, most of whom have extensive cicatricial contraction of the conjunctiva and more or less impairment of vision as a result of pannus and ulcers, and some of the more malignant cases in younger people and children, the question of tarsectomy, or the combined excision of the transition folds and part of the thickened tarsus, deserves consideration.

Personally, I feel that such operations are indicated only in selected cases, cases that exhibit extensive thickening and infiltration of the tarsus and cul-de-sac or marked papillary hypertrophy of the tarsal conjunctiva. I do not believe that such operations should be considered as routine measures in treating the disease except in a limited number of selected cases. We have had some splendid results from tarsectomy in selected cases, also, in selected cases, good results from the combined excision of the fornix and tarsus. But our results have not all been good. We have had and seen others have some bad results following these operations, varying degrees of lagophthalmos being the most common. These operations, in my experience, do not cure trachoma unless the disease is confined mostly to the tissue excised, and we do not see many cases where it is limited to the upper conjunctiva, tarsi and retrotarsal folds.

There are now three patients in our eye hospital under treatment for trachoma who were tarsectomized two years ago. Two of them have extensive trachomatous infiltration of the conjunctiva of the lower lids and upper transition folds.

Just what per cent. of cases are actually cured I cannot say. The majority of our patients show varying degrees of improvement within a short time, two to four weeks, after treatment is begun. Within six months to a year, in the ordinary case, the symptoms mostly disappear. However, acute flareups in the so-called cured cases are common and one is constantly being impressed with the fact that treatment has been too desultory.

Very sincerely yours,
P. RICHARDS, M.D.

Fort Defiance,
Arizona.

On the Operative Treatment of Trachoma.

To the Editor of Archives of Ophthalmology.

Dear Sir:

There are many questions in eye surgery on which American and European ophthalmologists have different opinions. One of the questions which is far from being settled is the operative treatment of trachoma. I have learned during my trip in the United States that there are prominent eye surgeons who frequently perform the combined excision of Kuhnt. There was a period about sixteen years ago when this operation was favored by many European eye surgeons and was especially popular on account of the excellent early results. In Hungary, which was before the war a country with an unusually large trachoma material, this operation was soon given up on account of its very disappointing end results. We perform it now only in very exceptional cases, and then not for trachoma, but for tarso-conjunctival tumors or tuberculosis.

In trachoma we have healing with scar formation. The scar replacing the destroyed conjunctiva shrinks and finally the fornix almost totally disappears and the tarsal conjunctiva then continues directly into the bulbar conjunctiva. We can prevent this if the patient comes in time. Among the several mechanical treating methods we found that "abrasion" of follicles and of papillary hypertrophies, as it was recommended by Imre, Sr., more than thirty years ago, gives quicker and more lasting results than any others. After doubly everting the eyelid with a broad double hook, or much more easily with a broad Desmarres spoon, the whole conjunctiva of the upper or of the lower lid is stretched out before us. We scrape off the follicles thoroughly, opening the covering membrane of those which are deeper set. While doing the abrasion we hold the convex sacrificator perpendicularly to the surface of the conjunctiva. Thus we clean the tarsal conjunctiva, fornix and semilunar fold. We also scarify the papillary hypertrophies. The instrument slips over the healthy parts of the conjunctiva without injury. Silver nitrate is used afterwards, but we take care not to use it too long and only when indicated. If there are new follicles four to five weeks later we remove them by the same method. This is by far

superior to expression, because we see the field and do not injure, tear or squeeze healthy tissues.

If the tarsus is strongly infiltrated, thickened or has already lost its natural shape by scar tissue, we perform the simple tarsectomy, sacrificing a narrow strip of conjunctiva already ruined in the region of the subtarsal sulcus. A great part of the tarsal conjunctiva will serve to cover the wound after some undermining towards the fornix. Thus we free the patient from his troublesome tarsus and we will find that by loosening the conjunctiva from the tarsus the hypertrophies and imbedded follicles soon disappear, and the diseased conjunctiva will become thin and pale.

But if we make a broad excision as we used to do when performing the combined excision, the part in the fornix is just enough to cover the wound surface after thorough undermining; this will shrink considerably when the scar tissue caused by the operation shrinks. It will become even shorter if, as in many cases, the pathological process is not checked in the fornix by the operation. This may seem to contradict what I have just said about the good influence which the undermining of the tarsal conjunctiva exerts. There it helps, because we leave only a very thin layer without nourishing subconjunctival tissue, whereas, the fornix remains in connection with the loose subconjunctival layer.

Kuhnt restricted the indications of this operation to those cases in which the trachomatous changes end in a sharp line and where the conjunctiva fornicis to be used for covering the tarsal defect can be considered healthy. Even in such cases relapses appear and the treatment will meet with great difficulties because we cannot properly reach the diseased regions.

After a large experience on a rich trachoma material we found the abrasion to be the most reliable mechanical way of treatment. We often perform tarsectomy on more advanced cases; even in those we make abrasion if there are follicles. We perform lid margin operations, like margino-plasty, Spencer-Watson operation, electrolytic epilation and tarsoplasty of Blaskovics, but the combined excision has lost favor as it is too radical and not reliable enough. Very sincerely yours,

Dr. Joseph Imre,
Pécs, Hungary.

Technique of Intracapsular Extraction.

January 26, 1927.

The Editor.

Sir:

Dr. L. L. Bull's letter in your issue of January this year raises many points of interest to your readers with which I wish to deal.

First, the matter of priority; I care little about it, but if the *Indian Medical Gazette* of 1906 is referred to, it will be seen that I had tried pressure with the squint hook over the sclerotic in an endeavor to make the hard lens tumble. Unfortunately, I had two cases of choroidal hemorrhage the first day, which I erroneously attributed to the method, and in consequence I abandoned it. It is clear that it is the logical outcome, at which someone must some day have arrived, intracapsular extraction having been established, and as a matter of fact in the winter of 1926–7 I found that other extensive operators were working towards this end quite independently of Dr. Mathra Das, though they had not yet got quite as far as he had.

With reference to the details of Mathra Das' technique, I saw him at work and find Dr. Bull's description of it very inadequate. He says that his incision is 180° in extent. As a matter of fact it is 240° for the big Morgagnian ending in the sclero-cornea, 220° for the hard cataract ending well forward in the cornea. Dr. Bull's *2mm* is rather an underestimate. He uses no spoon counter pressure over the wound in the hard cataract. His left hand is occupied to outward appearance solely in controlling the orbicularis, the index finger round the shaft of the squint hook, and the thumb retracting the eyebrow. He is surrounded by a horde of spectators, 20 or more in number, all blocking one another's view, not one in 500 of whom will notice what his left thumb is really doing. When I was there, a young lady said to him: "I often see you pressing with your thumb." His answer was a grunt. He operates without one word of explanation of anything. His pupils hold the instrument, but his hands are on it, too, and it is his hands which operate it and express the lens. Under these circumstances his pupils cannot learn

anything of what is happening. His assistants nod their heads gravely. "It is all a trick of the fingers, the Babu Sahib is not an educated man and does not pretend to be able to teach."

The forward incision is plainly designed to prevent the upper edge of the lens coming out first, with consequent escape of vitreous, but this is frequently not sufficient, as anyone who operates according to Dr. Bull's description will quickly find. The left hand thumb pressure is then called into play to supplement this forward incision, but is apparently not intended to be seen or understood.

I have not one hard word to say of Dr. Mathra Das. He was one of my earliest and most successful pupils, of whom the Punjab contains many, and deserves all credit for the progress he has made in this important field. But, at the same time, I have no desire to claim precedence for an operation implying an incision of from 220° to 240° and ending forward in the cornea. What I do is quite different. I use a sclero-corneal incision of 180°, and counter-pressure with a spoon over the wound in all cases of hard cataract. The squint hook must be applied well below the sclero-cornea, 5*mm* or more from it in the first instance. Less is insufficient, for it is liable to be in front of the lower edge of a large lens, and to achieve its purpose properly the pressure must be behind it.

Finally, I would like to know whether Dr. Bull is himself practising the operation, and why he has waited for four years before letting the world know about it. It will be clear from what I have written how much he really saw of what was going on. Is it that he did not appreciate the importance of what he saw until he read my article and that had it been left to him to report the matter we should never have heard of it?

HENRY SMITH, C.I.E., Lt. Col., I.M.S.

54 PARLIAMENT ST.,
LONDON.

XVIIth ANNUAL MEETING
OF THE
OXFORD OPHTHALMOLOGICAL CONGRESS.

Master, PHILIP H. ADAMS.
Hon. Treasurer, ROBERT J. COULTER.
Hon. Secretary, BERNARD CRIDLAND, Salisbury House, Wolverhampton.
Founder, the late ROBERT W. DOYNE.

February, 1927.

PRELIMINARY NOTICE.

The Oxford Ophthalmological Congress will assemble at Keble College on the evening of Wednesday, July 6th next, and the Meeting will be held on Thursday, July 7th, Friday, July 8th, and Saturday, July 9th.

On the morning of Thursday, July 7th, a discussion will be held on "The Results of the Operative Treatment of Glaucoma," to be opened by DR. WILLIAM H. WILMER, Ophthalmologist-in-Chief to the Johns Hopkins Hospital, Baltimore, Md., U.S.A.

Members intending to take part in the Discussion are requested to kindly send in their names to the Hon. Secretary at their early convenience.

The Doyne Memorial Lecture will be delivered on the morning of Friday, July 8th, by Professor K. K. K. Lundsgaard of Copenhagen, the subject being "The Pneumococcus in Connection with Ophthalmology."

It is again proposed to devote one afternoon entirely to

demonstrations in the Scientific and Commercial Museums and it is hoped that members will do their best to bring forward novelties of any kind, *e.g.* pathological specimens, operations, apparatus, etc.

It will be appreciated if those who desire to contribute either papers or demonstrations will kindly notify the Hon: Secretary at their early convenience.

The Official Dinner of the Congress will take place on the evening of Thursday, July 7th, in the Hall of Keble College.

A General Meeting will be held during the Congress at a time of which due notice will be given in the final programme.

BERNARD CRIDLAND,
Hon. Secretary.

Salisbury House,
Wolverhampton.